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Cancer Risk from Outdoor Exposure to Air Toxics. Volume 1

Pacific Environmental Services, Inc., Durham, NC

Prepared for:

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16. ABSTRACT

This report presents an analysis of cancer risks in the United States from outdoor exposures to airborne toxic pollutants. It is intended to provide updated information to suggest priorities for air toxics control. This study is an update of an EPA report issued in 1985 entitled The Air Toxics Problem in the United States: An Analysis of Cancer Risks for Selected Pollutants (EPA-450/1-85-001, May 1985), known as the "Six-Month Study."

This analysis is based primarily on information derived from recent studies and reports. Results are expressed as cancer risk from individual pollutants and source categories in terms of excess lifetime individual cancer risks and nationwide annual cancer cases.

Health risks due to indoor exposure and noncancer health effects resulting from outdoor exposure are not included in this analysis, but are addressed in separate studies.

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CANCER RISK FROM OUTDOOR EXPOSURE TO AIR TOXICS

Volume I

Final Report

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CANCER RISK FROM OUTDOOR EXPOSURE TO AIR TOXICS

VOLUME I

FINAL REPORT

U.S. Environmental Protection Agency
Office of Air Quality Planning and Standards
Research Triangle Park, North Carolina 27711

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GLOSSARY

Ambient (air) monitoring. The collection of ambient air samples and the analysis thereof for air pollutant concentrations.

Acute exposure. One or a series of short-term exposures generally lasting less than 24 hours.

Additivity. A pharmacologic or toxicologic interaction in which the combined effect of two or more chemicals is approximately equal to the sum of the effect of each chemical alone. (Compare with: antagonism, synergism.)

Adverse effect. A biochemical change, functional impairment, or pathological lesion that either singly or in combination adversely affects the performance of the whole organism, or reduces an organism's ability to respond to an additional environmental challenge.

Aggregate risk. The sum of individual increased risks of an adverse health effect in an exposed population.

Annual incidence. The number of new cases of a disease occurring or predicted to occur in a population over a year.

Antagonism. A pharmacologic or toxicologic interaction in which the combined effect of two chemicals is *less than* the sum of the effect of each chemical alone; the chemicals either interfere with each other's actions, or one interferes with the action of the other. (Compare with: additivity, synergism.)

Areawide average individual risk. Average individual risk to everyone in an area (but not necessarily the actual risk to anyone). May be computed by dividing lifetime aggregate incidence by the population within the area.

Areawide incidence. Incidence over a broad area, such as a city or county, rather than at a particular location, such as an individual grid cell.

Background. A term used in dispersion modeling representing the contribution to ambient concentrations from sources not specifically modeled in the analysis, including natural and manmade sources.

Bioassay. A test conducted in living organisms to determine the hazard of potency of a chemical by its effect on animals, isolated tissues, or microorganisms.

Box model. A simplified modeling technique that assumes uniform emissions within an urban area and uniformly mixed concentrations within a specified mixing depth.

Cancer. A malignant new growth. Cancers are divided into two broad categories: carcinoma and sarcoma.

Carcinogenic. Able to produce malignant tumor growth. Operationally most benign tumors are usually included also.

Carcinogenic process. A series of stages at the cellular level after which cancer will develop in an organism. Some believe there are at least 3 stages. initiation, promotion, and progression. While hypothesized as staged process, little is known about specific mechanisms of action.

Chronic exposure. Long-term exposure usually lasting six months to a lifetime.

Comparative potency factor. A cancer unit risk factor for a complex substance or mixture that is extrapolated from human risk data for a reference substance and the ratio of short term bioassay responses of the complex substance to the reference substance. The EPA is developing comparative potency factors for various classes of POM.

Confidence limit. The confidence interval is a range of values that has a specified probability (e.g., 95 percent) of containing a given parameter or characteristic. The confidence limit referees to the upper value of the range (e.g., upper confidence limit).

Criteria pollutants. Pollutants defined pursuant to Section 108 of the Clean Air Act and for which national ambient air quality standards are prescribed. Current criteria pollutants include particulate matter, SO_x, NO_x, ozone, CO and lead.

Dispersion modeling. A means of estimating ambient concentrations at locations (receptors) downwind of a source, or an array of sources, based on emission rates, release specifications and meteorological factors such as wind speed, wind direction, atmospheric stability, mixing height and ambient temperature.

Dose-response relationship. A relationship between: (1) the dose, often actually based on "administered dose" (i.e., exposure) rather than absorbed dose, and (2) the extent of toxic injury produced by that chemical. Response can be expressed either as the severity of injury or proportion of exposed subjects affected. A dose-response assessment is one of the four steps in a risk assessment.

Excess risk. An increased risk of disease above the normal background rate.

Exposure. Contact of an organism with a chemical, physical, or biological agent. Exposure is quantified as the amount of the agent available at the exchange boundaries of the organism (e.g., skin, lungs, digestive tract) and available for absorption.

Exposure assessment. Measurement or estimation of the magnitude, frequency, duration and route of exposure of animals or ecological components to substances in the environment. The exposure assessment also describes the nature of exposure and the size and nature of the exposed populations, and is one of four steps in risk assessment.

Human Exposure Model (HEM). A mathematical model used in exposure assessments for toxic air pollutants to quantify the number of people exposed to pollutants emitted by stationary sources and the pollutant concentrations they are exposed to. Input data include plant characteristics such as location, emission, parameters, etc. as well as Bureau of Census data used in the estimation of persons exposed and appropriate meteorological data.

Incidence. The number of new cases of a disease within a specified time period. It is frequently presented as the number of new cases per 1,000, 10,000, or 100,000. The incidence rate is a direct estimate of the probability or risk of developing a disease during a specified time period.

Individual risk. The increased risk for a person exposed to a specific concentration of a toxicant. May be expressed as a lifetime individual risk or as an annual individual risk, the latter usually computed as 1/70 of the lifetime risk.

Lifetime. Covering the lifespan of an organism (generally considered 70 years for humans).

Limited evidence. According to the USEPA carcinogen risk assessment guidelines, limited evidence is a collections of facts and accepted scientific inferences that suggests the agent may be causing an effect but the suggestion is not strong enough to be an established fact.

Lowest-observed-adverse-effect level (LOAEL). The lowest dose or exposure level of a chemical in a study at which there is a statistically or biologically significant increase in the frequency or severity of an adverse effect in the exposed population as compared with an appropriate, unexpected control group.

Lowest-observed effect level (LOEL). In a study, the lowest dose or exposure level at which a statistically or biologically significant effect is observed in the exposed population compared with an appropriate unexposed control group.

Malignant. A condition of a neoplasm (tumor) in which it has escaped normal growth regulation and has demonstrated the ability to invade local or distance structures, thereby disrupting the normal architecture or functional relationship of the tissue system.

Maximum individual risk (MIR). The increased risk for a person exposed to the highest measured or predicted concentration of a toxicant.

Maximum likelihood estimate (MLE). A statistical best estimate of the value of a parameter from a given data set.

Mobile source. Any motorized vehicle, such as cars, trucks, airplanes, trains. Sometimes refers specifically to highway vehicle sources.

Monitoring. The collection and analysis of ambient air samples. Sometimes refers specifically to just sampling and not to analysis. Can also refer to source (stack) sampling.

Motor vehicle. On-road or off-road cars, trucks or motorcycles.

Multistage model. A mathematical function used to extrapolate the probability of incidence of disease from a bioassay in animals using high doses, to that expected to be observed at the low doses that are likely to be found in chronic human exposure. This model is commonly used in quantitative carcinogenic risk assessments where the chemical agent is assumed to be a complete carcinogen and the risk is assumed to be proportional to the dose in the low region.

Mutagenic. Ability to cause a permanent change in the structure of DNA. More specific than, but often used interchangeably with, genotoxic.

Noncancer risk. Risk of a health effect other than cancer.

Nonthreshold toxicant. An agent considered to produce a toxic effect from any dose; any level of exposure is deemed to involve some risk. Usually used only in regard to carcinogenesis.

Nontraditional sources. Sources not usually included in an emission inventory, such as wastewater treatment plants, groundwater aeration facilities, hazardous waste combustors, landfills, which are air emitters due to intermedia transfer from water or solid waste.

No-observed-adverse-effect level (NOAEL). The highest experimental dose at which there is no statistically or biologically significant increases in frequency or severity of adverse health effects, as seen in the exposed population compared with an appropriate, unexposed population. Effects may be produced at this level, but they are not considered to be adverse.

No-observed-effect level (NOEL). The highest experiment dose at which there is no statistically or biologically significant increases in frequency or severity of toxic effects seen in the exposed compared with an appropriate, unexposed population.

Normalized modeling. Modeling of unit weights (e.g., 1 Mg/yr) of emissions from each source, rather than modeling of actual emissions, and displaying incremental receptor concentrations or receptor coefficients. Thereafter, the resulting normalized receptor coefficients are adjusted by actual emission rates to simulate different emission scenarios rather than re-running the model over and over with different emissions totals. This process assumes linearity between emissions and modeled ambient air concentrations, which does not always hold if stack and exhaust parameters change.

Photochemically formed pollutant. A secondarily formed pollutant due to atmospheric photochemistry. Some examples are formaldehyde and PAN.

Potency. A comparative expression of chemical or drug activity measured in terms of the relationship between the incidence or intensity of a particular effect and the associated dose of a chemical, to a given or implied standard or reference.

Receptor. A particular point in space where a monitor is located or where an exposure or risk is modeled.

Receptor grid. An array of receptors. Generally synonymous with network.

Receptor modeling. A technique for inferring source culpability at a receptor(s) by analysis of the ambient sample composition. There are various receptor models employing microscopic and chemical methods for analysis.

Reference dose (RfD). An estimate (with uncertainty spanning perhaps an order of magnitude) of the daily exposure to the human population (including sensitive subpopulations) that is likely to be without deleterious effects during a lifetime. The RfD is reported in units of mg of substance/kg body weight/day for oral exposures, or mg of substance/m^3 of air breathed for inhalation exposures.

Risk. The probability of injury, disease, or death under specific circumstances. In quantitative terms, risk is expressed in values ranging from zero (representing the certainty that harm will not occur) to one (representing the certainty that harm will occur).

Risk assessment. The scientific activity of evaluating the toxic properties of a chemical and the conditions of human exposure to it in order both to ascertain the likelihood that exposed humans will be adversely affected, and to characterize the nature of the effects that they may experience. May contain some or all of the following four steps:

> **Hazard identification**. The determination of whether a particular chemical is or is not causally linked to particular health effect(s).
>
> **Dose-response assessment**. The determination of the relation between the magnitude of exposure and the probability of occurrence of the health effects in question.
>
> **Exposure assessment**. The determination of the extent of human exposure.
>
> **Risk characterization**. The description of the nature and often the magnitude of human risk, including attendant uncertainty.

Risk characterization. The final step of a risk assessment, which is a description of the nature and often the magnitude of human risk, including attendant uncertainty.

Risk management. The decision-making process that uses the results of risk assessment to produce a decision about environmental action. Risk management includes consideration of technical, scientific, social, economic, and political information.

Route of exposure. The means by which toxic agents gain access to an organism (e.g., ingestion, inhalation, dermal exposure, intravenous, subcutaneous, intramuscular, intraperitoneal administration).

Scoping study. Also known as screening study. An assessment of analysis using tentative or preliminary data whose results are not accepted as absolute indicators of risk or exposures, but rather, are taken as an indication of the relative importance of various sources, pollutants and control measures. Most urban air toxics assessments conducted to date have been considered to be scoping studies, useful for pointing out where more detailed work is needed prior to regulation.

Species profile. A set of apportioning factors that allow one to subdivided VOC or PM emission totals into individual chemicals or chemical classes. Generally, species profiles are multiplicative in nature.

Subchronic exposure. Exposure to a substance spanning approximately 10 percent of the lifetime of organism.

Synergism. A pharmacologic or toxicologic interaction in which the combined effect of two or more chemicals is greater than the sum of the effect of each chemical alone. (Compare with: additivity, antagonism.)

Threshold Limit Value (TLV). The concentration of a substance below which no adverse health effects are expected to occur for workers assuming exposure for 8 hours per day, 40 hours per week. TLVs are published by the American Conference of Governmental Industrial Hygienists (ACGIH). This listing may be useful in identifying substances used in the workplace and having the potential to be emitted into the ambient air.

Threshold toxicant. A substance showing an apparent level of effect that is a minimally effective dose, above which a response occurs; below that dose no response is expected.

Transformation. The conversion, through chemical or physical processes, of one compound or several compounds into other compounds as a result of aging and irradiation in the atmosphere.

Transport. The movement of pollutants by wind flow. Transport is characterized for modeling purposes by wind speed and wind direction.

Unit cancer risk. A measure of the probability of an individual's developing cancer as a result of exposure to a specified unit ambient concentration. For example, an inhalation unit cancer risk of 3.0 x 10^{-4} near a point source implies that if 10,000 people breathe a given concentration of a carcinogenic agent (e.g., 1 $\mu g/m^3$) for 70 years, three of the 10,000 will develop cancer as a result of this exposure. In water the exposure unit is usually 1 $\mu g/l$, while in air it is 1 $\mu g/m^3$.

Weight-of-evidence. The extent to which the available biomedical data support the hypothesis that a substance causes an effect in humans. For example, the following factors increase the weight-of-evidence that a chemical poses a hazard to humans; an increase in the number of tissue sites affected by the agent; an increase in the number of animal species, strains, sexes, and number of experiments and doses showing a response; the occurrence of a clear-cut dose-response relationship as well as a high level of statistical significance in the occurrence of the adverse effect in treated subjects compared with untreated controls; a dose related shortening of the time of occurrence of the adverse effect; etc.

ACRONYMS

ATERIS	Air Toxic Exposure and Risk Information System
BaP	Benzo(a)pyrene
BID	Background information document
CAG	Carcinogen Assessment Group
CDD/CDF	Chlorinated dibenzo-p-dioxins and chlorinated dibenzofurans
DOE	Department of Energy
EDB	Ethylene dibromide
EDC	Ethylene dichloride
EPA	Environmental Protection Agency
HEM	Human exposure model
IACP	Integrated Air Cancer Program
IEMP	Integrated Environmental Management Project
LOAEL	Lowest-observed-adverse-effect level
LOD	Limit of detection
MEI	Maximum exposed individual
MIR	Maximum individual risk
MLE	Maximum likelihood estimate
MWC	Municipal waste combustor
NESHAP	National emission standard for hazardous air pollutants
NRC	Nuclear Regulatory Commission
OAQPS	Office of Air Quality Planning and Standards
PCB	Polychlorinated biphenyl
PIC	Products of incomplete combustion
POHC	Principal organic hazardous constituents
POM	Polycyclic organic matter

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POTW	Publicly owned treatment works
PUL	Plausible upper limit
RCRA	Resource Conservation and Recovery Act
SARA	Superfund Amendments and Reauthorization Act
SHED	SAI Human Exposure Dosage Model
STAPPA/ALAPCO	State and Territorial Air Pollution Program Administrators and the Association of Local Air Pollution Control Officials
STB	Science and Technology Branch
TCCD	Tetrachlorinated dibenzodioxin
TSDF	Treatment, storage, and disposal facilities (for hazardous waste)
TSP	Total suspended particulates
VOC	Volatile organic compound
$\mu g/m^3$	microgram per cubic meter
g/mile	grams per mile

EXECUTIVE SUMMARY

This report presents an analysis of cancer risks in the United States from outdoor exposures to airborne toxic pollutants. It is intended to provide updated information to suggest priorities for air toxics control. This study is an update of an EPA report issued in 1985 entitled The Air Toxics Problem in the United States: An Analysis of Cancer Risks for Selected Pollutants (EPA-450/1-85-001, May 1985), known as the "Six-Month Study."

This analysis is based primarily on information derived from recent studies and reports. Results are expressed as cancer risk from individual pollutants and source categories in terms of excess lifetime individual cancer risks[1] and nationwide annual cancer cases.

Health risks due to indoor exposure and noncancer health effects resulting from outdoor exposure are not included in this analysis, but are addressed in separate studies.[2] Risks from indoor exposures to certain pollutants can be significant because of higher indoor concentrations and the fact that most people spend much of their time indoors. Noncancer risks from outdoor exposure also may be significant, but more information is needed to adequately quantify these risks.

About 90 toxic air pollutants and 60 source categories were addressed in one or more of the studies examined. Additional risks associated with other pollutants and sources are not characterized. Of particular concern is the absence of information on pollutants secondarily formed in the atmosphere. Only one (formaldehyde) is considered in this analysis.

Significant uncertainties are associated with estimating risk. These are due to both data limitations and assumptions inherent in our current risk assessment methodology and the methodology required to combine and extrapolate information from individual studies to develop national estimates.

Assumptions about cancer potencies of various chemicals or chemical mixtures are generally considered to overestimate the risk, as do some assumptions about exposures. Uncertainties such as those due to missing pollutants, uncharacterized sources, long-range transport of pollutants, and pollutant transformation in the atmosphere will underestimate the risk.

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[1] "Lifetime individual risk" is a measure of the probability that an individual will develop cancer as a result of exposure to an air pollutant over a lifetime (i.e., a 70-year period).

[2] See Report to Congress on Indoor Air Quality (EPA-400/1-89-001, August, 1989) for current estimates of cancer public health risks from exposure to indoor air toxics. EPA also is evaluating the noncancer public health risks resulting from short-term and long-term outdoor exposures to toxic air pollutants. This latter study is discussed in Appendix C of this report.

Major findings on national cancer incidence and lifetime individual risk, which are subject to uncertainties and data limitations as noted above, are highlighted below.

Cancer Incidence

- Based on the pollutants and source categories examined, total excess cancer cases were estimated to be between 1,700 and 2,700 per year nationwide. This is equivalent to between 7 and 11 cancer cases per year per million population.
- Of the approximately 90 pollutants evaluated, 12 accounted for over 90% of total annual cancer incidence. Of these, PIC (products of incomplete combustion) were responsible for about 35% of the total. Other major contributors include 1,3-butadiene, hexavalent chromium, benzene, formaldehyde, and chloroform.
- Motor vehicles accounted for almost 60% of total cancer incidence. Other area sources accounted for approximately 15% of the total. Point sources accounted for the remaining 25% of the total annual cancer incidence.

Lifetime Individual Risk

- Maximum lifetime individual risks exceeding 10^{-4} (exceeding 1 chance in 10,000 of contracting cancer) from multi-pollutant exposures were reported in almost all studies. Risks of 10^{-3} or greater from individual pollutants were reported adjacent to various types of sources.
- The relative contribution of pollutants and sources to risk in a specific urban area can vary significantly. However, the areawide lifetime individual risks in urban areas from the combined exposure to many pollutants generally are in the 10^{-4} range, but varied from 10^{-5} to 10^{-3}. These levels result from exposure to emissions from mobile and stationary sources combined.

The numerical estimates presented in this report should be viewed only as rough indications of the potential for cancer risk caused by a limited group of pollutants found in the ambient air. Many of the risks cited in this report are almost certainly inaccurate in an absolute sense. The best use of the risk estimates is in describing the broad nature of cancer risk posed by these toxic air pollutants and by making relative comparisons of risks between pollutants and sources.

The technical approach for this study, including a description of the methodology and a discussion of uncertainties and assumptions, is presented in the next section. Additional information on major findings is provided under Results, and a comparison with the findings of the

1985 Six-Month Study is presented under Comparison With 1985 Six-Month Study.

TECHNICAL APPROACH

Sources of Information

This study is based on information contained in 10 area-specific or national air quality based risk-related reports on air toxics, 14 EPA source category and pollutant-specific studies, risk assessments performed for the development of National Emission Standards for Hazardous Air Pollutants (NESHAP), and source specific risk data contained in the EPA Air Toxic Exposure and Risk Information System (ATERIS) data base. These reports and studies are described in Chapter 2 of this report. They represent a much larger data base and more comprehensive coverage than used for the 1985 Six-Month Study.

Additional information on air toxics emissions data is being collected under Title III of the Superfund Amendments and Reauthorization Act (SARA). However, in their present form, these data can not be used to estimate risks. Therefore, this study does not present risk estimates based on the SARA Title III emissions data.

Methodology

Estimates of annual cancer incidence were derived by first developing estimates of the annual cancer cases per million population for each pollutant/source category combination (e.g., 1,3-butadiene emissions from mobile sources) reported in the data sources. These were modified as necessary to reflect updated unit risk and emission factors. Estimates of total nationwide annual incidence then were calculated, in most instances, by multiplying the annual cancer cases per million population by the total U.S. population and then summing across all pollutant/source categories. Lifetime individual cancer risk estimates either were obtained directly from each study or modified based on updated information.

Because studies were of varying quality and most were concerned with specific geographic areas, source categories, and/or pollutants, a number of factors had to be examined to evaluate study results before they could be combined and extrapolated to obtain national cancer incidence estimates. These include the geographic scope of the study, source category definitions, unit risk factors, method of estimating ambient concentrations (modeled vs. monitored), and emission estimates. These factors are discussed below.

Geographic Scope of Studies. Cancer rates for a pollutant and source category were extrapolated to nationwide estimates based on the geographic scope of each study examined. Most pollutant/source categories were included in at least one study that was nationwide in scope and this permitted a direct extrapolation to total nationwide estimates.

A few pollutants and source categories were included only in a study of limited geographic scope. In such instances, it was determined whether the pollutant/source category might be unusually concentrated in the area studied or was fairly common across the United States. This information was used to determine how study results could be extrapolated to obtain total nationwide estimates.

Source Category Definitions. Source category definitions in each study were examined to minimize the possibility of double-counting. This was especially difficult for the heating/combustion source category because the various studies used different terminology and not all reports clearly indicated what was or was not covered.

Unit Risk Factors. The unit risk factor is defined as an estimate of the probability that an individual will develop cancer when exposed to a pollutant at an ambient concentration of one microgram per cubic meter ($\mu g/m^3$) for 70 years. These are either upper-bound values or maximum likelihood values.[3] The estimate of cancer risk for each pollutant, considering the unit risk factor alone, is conservative; that is, while the actual risk may be higher, it is more likely to be lower and may even be as low as zero. The weight-of-evidence that a pollutant causes cancer varies from proven human carcinogen (e.g., benzene) to probable human carcinogen (e.g., 1,3-butadiene) to possible human carcinogen (e.g., vinylidene chloride). All were included in this analysis as carcinogens.

The cancer rates presented in the studies were updated, as necessary, based on common unit risk factors used by EPA. With one exception, this adjustment generally had little effect on the magnitude of the total risk estimated by the various studies. The exception was the South Coast study where the estimated cancer risk was 10 times higher than the adjusted estimate based on EPA factors.

Although the unit risk factors used in this report come from EPA studies, not all of them have been officially approved by EPA. In addition, many of the unit risk factors remain uncertain and are subject to change as further evidence of carcinogenicity is obtained. For many substances, this factor probably has the greatest potential for error in estimating cancer risk. This is a significant issue and affects pollutants such as formaldehyde, vinyl chloride, products of incomplete combustion (PIC[4]), and diesel particulate (which is included with PIC).

Of particular concern are the unit risk factors for PIC mixtures since these mixtures are responsible for about one-third of the cancer cases estimated in this study. While many unit risk factors used in this study have been approved by EPA, PIC is an important exception.

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[3] "Maximum likelihood estimate" refers to a statistical best estimate of the risk.

[4] "PIC" is primarily composed of "polycyclic organic matter" (POM). Benzo(a)pyrene (BaP), which is used as a surrogate for PIC exposure, is a component of POM.

There are no current EPA-approved unit risk factors for these mixtures or for individual PIC components, although unapproved unit risk factors are available for some of the compounds (e.g., benzo(a)pyrene). A number of methods have been used to estimate the aggregate carcinogenic potency of various PIC mixtures. The values and method selected for this study were based on a review of ten EPA studies that included risk estimates for PIC. In view of the potentially high risks associated with PIC, there is a need for more thorough review to establish an EPA-approved risk methodology and unit risk factor(s) for PIC mixtures that can be used in future studies of this type.

Modeled vs. Measured Ambient Concentrations. Cancer risk estimates can be derived from either modeled or measured ambient concentrations. Each method has advantages and limitations. Both ambient and modeled estimates were given equal weight in estimating cancer risk unless there were clear reasons to prefer one estimate to another.

A limitation of dispersion models is the need for accurate estimates of pollutant emissions. A limitation of monitoring for many analyses is the difficulty of monitoring at enough locations to characterize the variability of ambient concentrations. This is true primarily for point source analyses. In urban areas where the object is to estimate average individual risk over a wide area, the specific location of the monitor may not be as critical. In this case, the use of measured ambient concentrations for risk estimation should provide more credible and reliable results than reliance on modeled estimates.

Modeling and monitoring produced similar risk estimates for some pollutants, such as for cadmium, methylene chloride, and trichloroethylene. For others, such as chloroform, ethylene dibromide, and formaldehyde, risk estimates based on measured ambient concentrations were greater than model-based estimates.

For formaldehyde, the difference in results probably is due to the fact that this pollutant is formed primarily in the atmosphere from other volatile organic compounds. Models are not yet available which can properly account for this, whereas ambient measurements do. The reasons for the different results for chloroform and ethylene dibromide are not clear, but a likely possibility is that the modeled results do not include all sources of emissions of these pollutants.

Emission Estimates. Modeled ambient concentrations, and therefore cancer risk estimates, are directly proportional to source emissions. The quality of emissions data can vary significantly. Three pollutants for which large uncertainties are associated with emissions estimates are dioxin, gasoline vapors, and hexavalent chromium. These uncertainties are recognized by reporting the risk from these pollutants as a range.

The uncertainty in dioxin emissions estimates is associated with emissions from hazardous waste treatment, storage, and disposal facilities (TSDFs). The range of emission estimates for gasoline vapors is due to the uncertainty as to the fraction of vapors that is carcinogenic.

With respect to chromium, only hexavalent chromium is known to be a carcinogen, but only total chromium is measured. Information has been developed on the percent of total chromium emissions that is hexavalent for specific emission sources, such as cooling towers, and this has been used in modeling studies. Studies based on measured ambient concentrations assume that a fraction of the measured chromium is hexavalent, but this is not well-defined.

Pollutants and Sources Not Evaluated

Although approximately 90 different toxic air pollutants and over 60 source categories were addressed in one or more of the studies used in this report, there are thousands of airborne chemicals that are potentially toxic, but have neither adequate exposure nor health effects data. Also, reliable quantitative emission estimates remain unavailable for many potentially important source categories. The lack of data for these pollutant and source categories could result in a significant underestimate of risk.

There also is a lack of information on risks associated with pollutants formed photochemically in the atmosphere (i.e., secondary formation). There is evidence that the mutagenicity of mixtures of some pollutants increases greatly as they undergo transformation in the atmosphere, but insufficient data are available to derive cancer risk estimates. Data on only one secondarily formed pollutant (formaldehyde) are included in this study.

Additive Risk

Total nationwide annual incidence was calculated by summing the risks for all pollutants and source categories. In addition, additive lifetime individual risks were obtained by summing risks for different pollutants at the same geographic location. This is the accepted approach and was used in all of the studies reviewed.

It should be noted that the assumption of additivity can lead to substantial errors in risk estimates if synergistic or antagonistic interactions occur. Although dose additivity has been shown to predict the acute toxicity of many mixtures of similar and dissimilar compounds, some marked exceptions have been identified. In some cases, risks would be greatly overestimated and, in other cases, greatly underestimated. The available data are insufficient for estimating the magnitude of these errors.

RESULTS

From the foregoing discussion, it is clear that there are numerous assumptions and significant uncertainties associated with the risk estimates in this study. In addition, potential sources of error are important to recognize and are discussed in detail in this report. In spite of these potential sources of error, it was concluded that point estimates would be a more useful way to compare risks among various pollutants and sources than by expressing broad ranges of risks. Nevertheless, for reasons discussed below, ranges were expressed for several pollutants.

Magnitude of the Problem

For the pollutants and source categories examined, the total nationwide cancer incidence due to outdoor concentrations of air toxics in the U.S. was estimated to range from approximately 1,700 to 2,700 excess cancer cases per year (see Table ES-1). This estimate is based on the most recent available unit risk factors and, in general, 1986 emissions data. It is roughly equivalent to between 7 and 11 annual cancer cases per million population (1986 population of 240 million).

The number of deaths resulting from these projected cancer cases is unknown. By way of comparison, the American Cancer Society has estimated total cancer deaths in the U.S. in 1989 to be 500,000.

The range of estimated excess cancer cases per year in this study is due primarily to the following uncertainties: (1) the unit risk factor for diesel particulate (which is included in the estimated cancer risk from PIC); (2) dioxin emissions from TSDFs; (3) the cancer-causing portion of gasoline vapors; and (4) the fraction of total chromium that is hexavalent.

Maximum lifetime individual risks of 1×10^{-4} (1 chance in 10,000 of contracting cancer) or greater were reported in almost all of the studies examined. Maximum lifetime individual risk levels exceeding 1×10^{-4} were reported for multi-pollutant exposures from such sources as major chemical manufacturers, waste oil incinerators, hazardous waste incinerators, municipal landfills, publicly owned treatment works (POTWs), and TSDFs.

Maximum individual risks of 1×10^{-4} or greater were reported adjacent to at least one source for each of 16 pollutants[5] included in the NESHAP/ATERIS data base. Twelve of these pollutants were estimated to be responsible for maximum individual risks of 1×10^{-3} or greater.

For the urban areas studied, areawide lifetime individual risks from all pollutants for point and area sources combined generally were in the 10^{-4} range, and ranged from 10^{-5} to 10^{-3}. Lifetime individual risks in four urban areas[6] due to multi-pollutant exposure (9 to 16 pollutants) ranged from 10^{-4} to 10^{-3} based on measured ambient concentrations. The contribution of specific area and point sources to

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[5] Acetaldehyde, acrylonitrile, arsenic, benzene, 1,3-butadiene, carbon tetrachloride, chloroform, hexavalent chromium, coke oven emissions, ethylene dichloride, epichlorohydrin, ethylene oxide, methylene chloride, p-dichlorobenzene, styrene, and vinylidene chloride.

[6] Los Angeles, Baton Rouge, Boston, and Chicago.

TABLE ES-1

SUMMARY OF ESTIMATED NATIONWIDE ANNUAL CANCER CASES BY POLLUTANT

POLLUTANT	EPA CLASSIFICATION[a]	ESTIMATED ANNUAL CANCER CASES
1. Acrylonitrile	B1	13
2. Arsenic	A	68
3. Asbestos	A	88
4. Benzene	A	181
5. 1,3-Butadiene	B2	266
6. Cadmium	B1	10
7. Carbon tetrachloride	B2	41
8. Chloroform	B2	115
9. Chromium (hexavalent)	A	147-265
10. Coke Oven Emissions	A	7
11. Dioxin	B2	2-125
12. Ethylene dibromide	B2	68
13. Ethylene dichloride	B2	45
14. Ethylene oxide	B1-B2	6
15. Formaldehyde	B1	124
16. Gasoline vapors	B2	19-76
17. Hexachlorobutadiene	C	9
18. Hydrazine	B2	6
19. Methylene chloride	B2	5
20. Perchloroethylene	B2	6
21. PIC	-[b]	438-1120
22. Radionuclides[c]	A	3
23. Radon[c]	A	2
24. Trichloroethylene	B2	7
25. Vinyl chloride	A	25
26. Vinylidene chloride	C	10
27. Miscellaneous[d]	-	15
Totals		1,726 - 2,706

NOTE: Values in this figure are not absolute predictions of cancer occurrence and are intended to be used in a relative sense only. The dose-response relationships and exposure assumptions have a conservative bias, but omissions due to uncharacterized pollutants (either directly emitted or secondarily formed) and emission sources, the long-range transport of pollutants, and the lack of knowledge of total risk from multi-pollutant exposures will offset this bias to an unknown extent.

FOOTNOTES TO TABLE ES-1

[a] For a discussion of how EPA evaluates suspect carcinogens and more information on these classifications, refer to "Guidelines for Carcinogen Risk Assessment" (51 Federal Register 33992).

The EPA classifications used in this report are:

A = proven human carcinogen; B = probable human carcinogen (B1 indicates limited evidence from human studies and sufficient evidence from animal studies; B2 indicates sufficient evidence from animal studies, but inadequate evidence from human studies); C = possible human carcinogen

[b] EPA has not developed a classification for the group of pollutants that compose products of incomplete combustion (PIC), although EPA has developed a classification for some components, such as benzo(a)pyrene (BaP), which is a B2 pollutant.

[c] From sources emitting significant amounts of radionuclides (and radon) to outdoor air. Does not include exposure to indoor concentrations of radon due to radon in soil gases entering homes through foundations and cellars.

[d] Includes approximately 68 other individual pollutants, primarily from the TSDF study and the Sewage Sludge Incinerator study.

these levels of risk generally could not be identified, but the relatively narrow range of the areawide lifetime individual risks in the urban areas studied suggests that other large urban populations may be subject to similar risk levels.

Nature of the Cancer Risk

Individual Pollutants. Available information suggests that 17[7] of the approximately 90 pollutants included in the data sources may each account for risks of at least 10 cancer cases per year. Of these, 13 pollutants[8] may each account for 40 or more cases per year. The relative contributions of these pollutants to the total annual cancer cases are shown in Figure ES-1.

The pollutants found in this study to be the primary contributors to annual cancer incidence also were frequently associated with high maximum individual risks. Other individual compounds, such as epichlorohydrin and styrene, which account for smaller aggregate cancer incidence, also are associated with high individual risks (greater than 1×10^{-4}).

Source Categories. Many types of sources contribute to aggregate incidence and lifetime individual risk. Figure ES-2 illustrates the relative contribution to total annual cancer incidence for each of the source categories evaluated.

On an individual source category basis, motor vehicles were the largest contributor to nationwide annual incidence, contributing approximately 58% of the total [including approximately 35% of the total contribution for which secondarily formed formaldehyde (shown as a separate category) is responsible]. Electroplating (6%) was another large contributor as a result of chromium emissions. Other major contributors are TSDFs (5%); woodsmoke (5%); asbestos, demolition (4%); gasoline marketing (3%); cooling towers (3%); and solvent use/degreasing (3%).

A significant portion of the cancer risk from most sources usually was due to a few pollutants, even where a source emitted many different pollutants. For example, over 70 pollutants were included in the analysis of hazardous waste combustors, but only two pollutants (cadmium and hexavalent chromium) were estimated to be responsible for almost 90 percent of the estimated cancer cases in this source category. Similarly, three pollutants (cadmium, hexavalent chromium, and arsenic) were responsible for almost 90 percent of the estimated cancer cases from hazardous waste boilers and furnaces.

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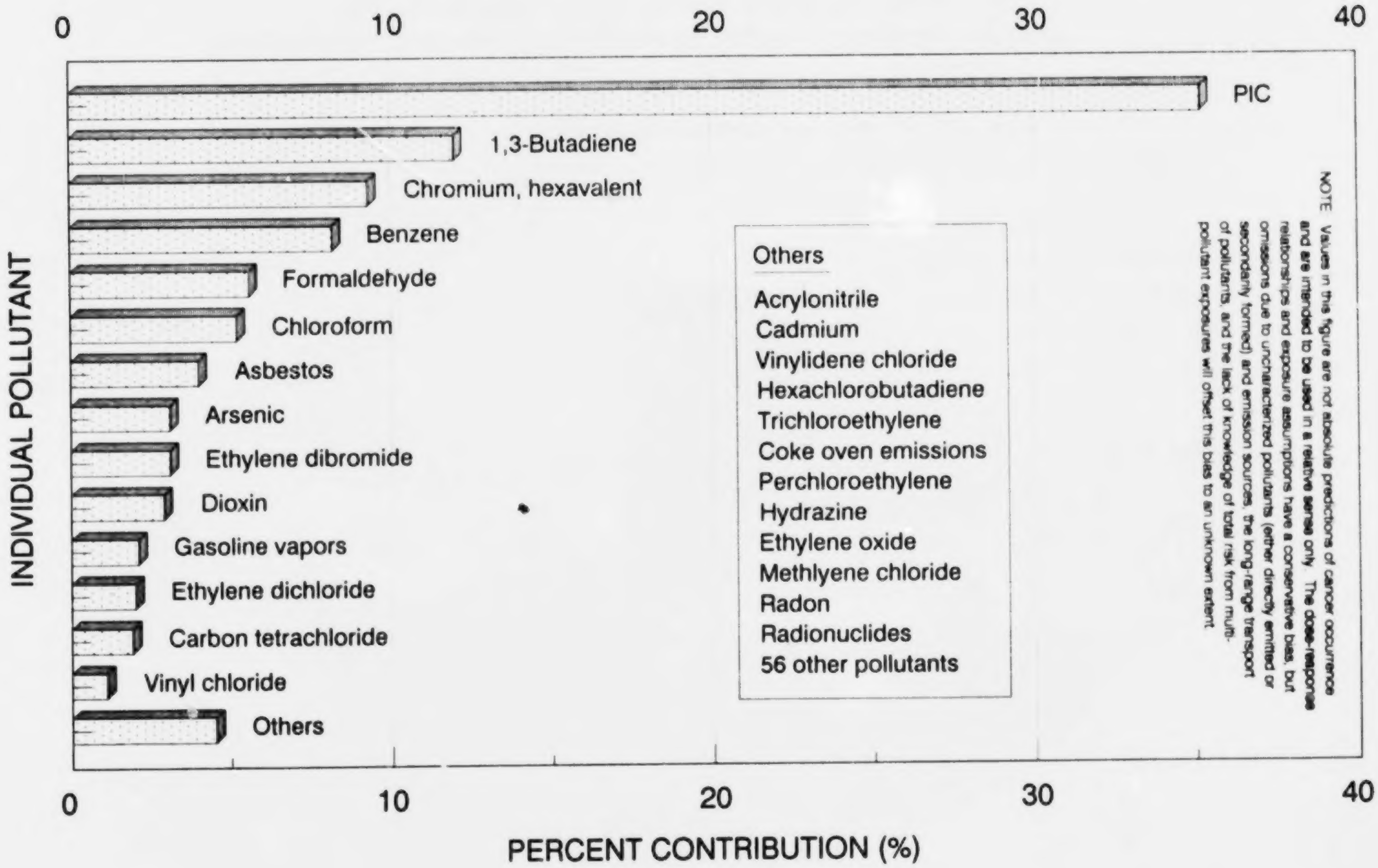
[7] Acrylonitrile, arsenic, asbestos, benzene, 1,3-butadiene, cadmium, carbon tetrachloride, chloroform, hexavalent chromium, dioxin, ethylene dibromide, ethylene dichloride, formaldehyde, gasoline vapor, PIC, trichloroethylene, and vinyl chloride.

[8] Arsenic, asbestos, benzene, 1,3-butadiene, carbon tetrachloride, chloroform, hexavalent chromium, dioxin, ethylene dibromide, ethylene dichloride, formaldehyde, gasoline vapor, and PIC.



NOTE: Values in this figure are not absolute predictions of cancer occurrence and are intended to be used in a relative sense only. The dose-response relationships and exposure assumptions have a conservative bias, but omissions due to uncharacterized pollutants (either directly emitted or secondarily formed) and emission sources, the long-range transport of pollutants, and the lack of knowledge of total risk from multi-pollutant exposures will offset this bias to an unknown extent.

Figure ES-1. Relative Contribution to Total Estimated Nationwide Cancer Cases Per Year, by Pollutant

NOTE: Values in this figure are not absolute predictions of cancer occurrence and are intended to be used in a relative sense only. The dose-response relationships and exposure assumptions have a conservative bias, but omissions due to uncharacterized pollutants (either directly emitted or secondarily formed) and emission sources, the long-range transport of pollutants, and the lack of knowledge of total risk from multi-pollutant exposures will offset this bias to an unknown extent.

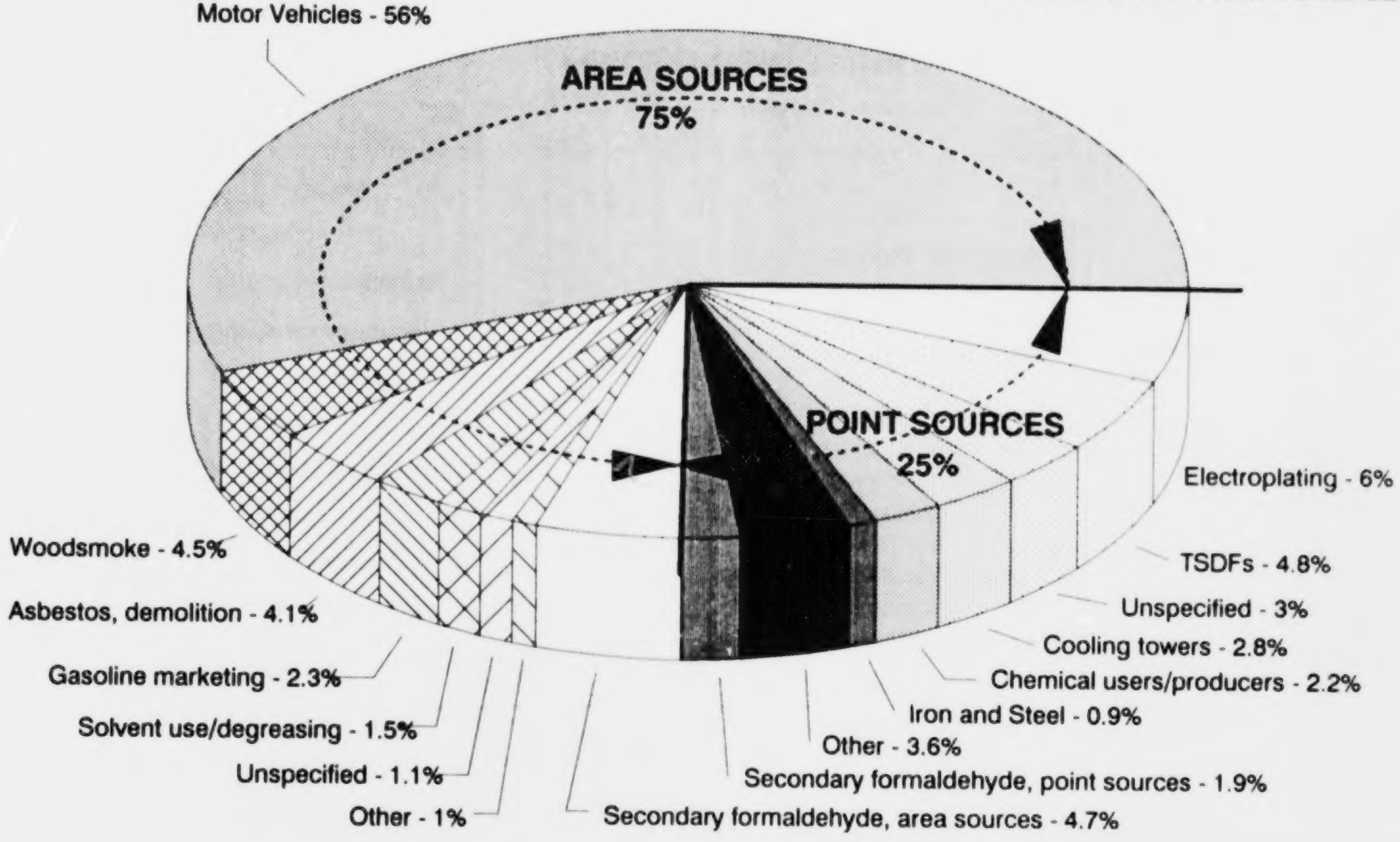


Figure ES-2. Relative Contribution by Source Categories to Total Estimated Nationwide Cancer Cases Per Year

Comparing aggregate source categories, mobile sources were estimated to contribute approximately 58 percent and stationary sources approximately 42 percent of the total annual incidence. Area sources were responsible for approximately 75 percent and point sources 25 percent of the total annual incidence associated with outdoor exposure to air toxics.

Geographic Variability. Ambient concentrations of individual air toxics vary on a city-to-city basis as well as on an intra-city basis. For the cities included in this study, the variation among cities ranged from a factor of 2 for benzene to almost 20 for chloroform. Similar variations were found within cities. Many factors could account for this. These include meteorological conditions, the location of sources relative to the population, and, where cancer risks were estimated from measured ambient concentrations, the location of the monitors.

COMPARISON WITH 1985 SIX-MONTH STUDY

The present study shows approximately 500 to 900 more cancer cases per year than reported in the 1985 Six-Month Study published in May, 1985. This apparent increase is due primarily to the inclusion of more pollutants, a better accounting of emission sources, and, in some cases, substantial increases in unit risk estimates.

The present study shows additive lifetime individual risks to be similar to those estimated in the 1985 Six-Month Study. However, the broader scope of the present study has identified additional source types (e.g., TSDFs, POTWs) that can cause high lifetime individual risks.

The individual compounds found in the present study to be the most important contributors to cancer risk are, for the most part, the same as those identified in the 1985 Six-Month Study. The most important addition is 1,3-butadiene. Dioxin also may be an important contributor, but the uncertainty associated primarily with estimates of dioxin exposure from TSDFs makes it difficult to conclude this at the present time. Several pollutants (asbestos, ethylene oxide, and trichloroethylene) appear to be somewhat less of a factor in terms of aggregate cancer risk, but not necessarily in terms of maximum individual risk.

The 1985 Six-Month Study found that area sources accounted for over 75 percent and point sources accounted for less than 25 percent of the total annual cancer incidence. This finding was essentially confirmed by the results of the present study. Findings in the present study on the geographic variability of risk also are reasonably consistent with those in the 1985 Six-Month Study.

1.0 INTRODUCTION

Background

The U.S. Environmental Protection Agency (EPA) initiated a broad "scoping" study in November, 1983, with a goal of gaining a better understanding of the size and causes of the health problems caused by outdoor exposure to air toxics. This broad scoping study, often referred to as the Six-Month Study[1], was published in May, 1985, and is hereafter referred to in this report as the 1985 Six-Month Study.

The objective of the 1985 Six-Month Study was to assess the magnitude and nature of the air toxics problem by developing quantitative estimates of the cancer risks posed by selected air pollutants and their sources from a national and regional perspective. It was designed to answer four basic questions:

1. What is the approximate magnitude of the air toxics problem, as measured by the estimated cancer risks associated with air pollution?
2. What is the nature of the air toxics problem; that is, what pollutants and sources appear to increase the incidence of cancer and what are their relative importance?
3. Does the cancer risk problem vary geographically and, if so, in what ways?
4. Are current air toxics data bases adequate for assessing the cancer risk from air toxics? If not, what are the significant data gaps?

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[1] Haemisegger, E. et. al. The Air Toxics Problem in the United States: An Analysis of Cancer Risks for Selected Pollutants. EPA-450/1-85-001. May 1985.

These questions were answered primarily by conducting three analyses to estimate cancer incidence (i.e., cancer cases per year) and lifetime individual risks.[2] One analysis estimated national exposure and risks from about 40 pollutants being considered for listing under Section 112 of the Clean Air Act.[3] The risks estimated in this analysis, which is referred to as the NESHAP (National Emission Standards for Hazardous Air Pollutants) Study, were national in scope and considered emissions obtained from traditional air pollution inventories. The emphasis of the NESHAP Study was on large point sources, but both mobile and area sources were also covered. The second analysis provided a more detailed estimate and analysis of exposure and risk in 35 counties for approximately 20 pollutants.[4] This second analysis, which is referred to as the 35-County Study, was designed to examine risk from air toxics on a more local perspective than the NESHAP Study. The analysis in the 35-County Study included sources not usually considered in previous studies, such as publicly owned treatment plants (POTWs) and waste oil combustors. The third analysis, which is referred to as the 1985 Ambient Air Quality Study, estimated cancer risks based on ambient air quality data for fourteen pollutants.[5] Quantitative risk assessments available from other EPA activities for asbestos, radionuclides, and gasoline marketing supplemented these three analyses in the 1985 Six-Month Study. Information available on several source categories for which data at that time were insufficient to perform a quantitative risk assessment were also analyzed and summarized in the study. The main conclusions reached in the 1985 Six-Month Study are summarized in Table 1-1.

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[2] "Lifetime individual risk" is a measure of probability that an individual will develop cancer as a result of exposure to the ambient concentration of an air pollutant over a lifetime (i.e., a 70-year period).

[3] Schell, R.M. Estimation of the Public Health Risks Associated with Exposure to Ambient Concentrations of 87 Substances. OAQPS, U.S. EPA, July 1984. Revised February 1985.

[4] Versar; American Management System, Inc. Hazardous Air Pollutants: An Exposure and Risk Assessment for 35 Counties. U.S. EPA Contract No. 68-01-6115, September 1984.

[5] Hunt, Bill, et. al. Estimated Cancer Incidence Rates from Selected Toxic Air Pollutants Using Ambient Data. U.S. EPA, revised March 1985.

Purpose of Current Study

The primary objective of the current study is to evaluate the magnitude and nature of the cancer problem associated with outdoor concentrations of air toxics in the United States. The magnitude of the cancer problem is addressed in terms of annual cancer incidence (i.e., the number of cancer cases per year nationwide) and lifetime individual risk (i.e., areawide and maximum individual risk[6]). The nature of the cancer problem is addressed primarily by examining the relative contributions of pollutants and sources to annual cancer incidence and the geographic variability of cancer risk and important contributors to that risk. In addition, the results of this study are compared with those of the 1985 Six-Month Study. Finally, while the current study does not include a reevaluation of EPA's air toxics control strategy, the study seeks to present information on the magnitude and nature of the air toxics problem due to outdoor exposure that may be used to help set priorities for the control of air toxics and to better define research and data needed to support a more effective control program.

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[6] "Areawide" individual risk refers to the average lifetime individual risk to everyone in an area. "Maximum" individual risk refers to the maximum level of risk to which a person could be exposed, and is located at a specific point within an area.

TABLE 1-1

MAIN CONCLUSIONS OF THE 1985 SIX-MONTH STUDY

- Nationwide annual cancer cases were estimated as 1,300 to 1,700 (5 to 7.4 cases of cancer per year per million population) for the 15 to 45 pollutants examined in each analysis.

- Maximum lifetime individual risks of 1×10^{-4} (1 in 10,000) or greater in the vicinity of major point sources were estimated for 21 pollutants, about half of those that were studied. Maximum lifetime individual risks of 1×10^{-3} (1 in 1,000) or greater were estimated for 13 pollutants.

- Additive lifetime individual risks in urban areas due to simultaneous exposure to 10 to 15 pollutants ranged from 1×10^{-3} to 1×10^{-4}. These risks, which were calculated from monitoring data, did not appear to be directly related to specific point sources. Instead, they represent a portion of total risks associated with the complex pollutant mixtures typical of urban ambient air.

- Thirteen specific pollutants[a] were identified as possibly important contributors to aggregate cancer cases from air toxics. Although little aggregate cancer incidence (less than 1 cancer case per year total) was found for 21 low production organic chemicals, some of these compounds appear to be associated with high individual risks. The low aggregate incidence for these compounds may be due in part to the lack of data concerning their emissions and toxicity.

- A wide variety of sources was found to contribute to cancer risk from air toxics, with combustion/incineration probably the largest single source of risk. Among this wide variety of sources were sources that have not historically been part of emission inventories, such as publicly owned treatment works (POTWs) and hazardous waste treatment, storage, and disposal facilities (TSDFs), which were found to possibly pose important risks in some locations.

- Both point sources (major industrial sources) and area sources (smaller sources that may be widespread across a given area, such as solvent usage and motor vehicles) appear to contribute significantly to cancer risk from air toxics. Large point sources tended to be associated with many high individual risks, while area sources appeared to be responsible for the majority of aggregate cancer cases.

- Where it could be analyzed, large city-to-city and neighborhood-to-neighborhood variation in pollutant levels and sources was found.

TABLE 1-1 (concluded)

MAIN CONCLUSIONS OF THE 1985 SIX-MONTH STUDY

- Major weaknesses and data gaps in the air toxics data bases at the Federal, State, and local levels made it impossible to accurately characterize most local air toxics problems. Problems identified with the few available air toxics data bases were inconsistencies and anomalies in the emission inventories, lack of sufficient data to develop population exposure estimates, and lack of compounds for which adequate health effects tests have been performed.
- EPA's criteria pollutant[b] program appears to have reduced air toxics levels. One analysis estimated the cancer rate from 16 air toxics in 1980 was less than half that for 1970 (6.8 vs. 17.5 cancer cases per year per million population).

SOURCE: Haemisegger, E. et. al. (1985) pp. 94-96.

[a] The thirteen pollutants were: chromium, arsenic, asbestos, products of incomplete combustion (PIC), formaldehyde, benzene, ethylene oxide, gasoline vapors, chloroform, carbon tetrachloride, perchloroethylene, trichloroethylene, and vinylidene chloride.

[b] EPA's criteria pollutants are: carbon monoxide, ozone, lead, total suspended particulate, oxides of nitrogen, and sulfur dioxide.

One of the key analyses in this study is based on combining the risk assessments from a number of information sources and developing an estimate of total cancer incidence from all of the pollutants and sources included in the various reports and studies. Ideally, this analysis would lead to a point estimate of total cancer incidence. While a point estimate seemed reasonable for a large number of individual pollutants, certain aspects of the risk assessment methodology for other pollutants did not allow for identifying a point estimate. For these other pollutants, only ranges could be identified. The analysis then tried to narrow the range as much as possible.

Existing information from various reports and studies available since the 1985 Six-Month Study was released has been gathered, organized, and evaluated in order to accomplish these objectives. Some of the quantitative estimates of risk used in this study to help identify high risk air toxics and sources come from studies that are part of the regulatory decision making process (e.g., background documents in support of NESHAPs under Section 112 of the Clean Air Act). Other quantitative risk estimates come from reports or studies that are of a general "scoping" nature and are not of the level of detail necessary for regulatory decisions. In addition, the risk estimates contained in these studies and reports are based on an uneven level of quality, which affects the certainty that one can attach to the risk estimates. For these reasons, the quantitative risk estimates can not be used to support regulatory decisions on source regulation. These results should, nevertheless, be useful in developing and evaluating air toxics control strategies and in establishing priorities within these strategies. Since there are limitations in most risk analyses, it is important for the reader using this report to consider the caveats and assumptions associated with the analyses in order to interpret and use the results properly.

Other Studies or Reports on Air Toxics

Health risk from air toxics encompasses both cancer and noncancer effects, and results from both indoor exposure as well as outdoor exposure to air toxics. This report examines cancer risk from outdoor concentrations of air toxics. The risk estimates presented in this study are associated with just one part of the total risk from air toxics (see Figure 1-1). Health risks from indoor exposure to air toxics and the noncancer risks from outdoor exposure to air toxics are the subjects of separate studies, which are discussed below. Also discussed are air toxics emissions data recently released under Title III of the Superfund Amendments and Reauthorization Act (SARA) of 1986.

Indoor Air Pollution

Under the Radon Gas and Indoor Air Quality Research Act of 1986 (Title IV of SARA), EPA is establishing a research program on all aspects of indoor air quality. As part of this program, EPA is seeking to identify high risk pollutant sources and characterize the exposures and health risks of various populations to those sources. Source categories that have been identified are: environmental tobacco smoke, combustion appliances, materials and furnishings, biological contaminants, consumer products (e.g., hair spray, paint solvents, cleaning fluids), outdoor sources (e.g., infiltration of radon, vehicle exhaust, pesticides), and nonionizing radiation. The indoor air program also addresses generic research activities. Generic research needs emphasize the concept of limiting total exposures and include development of standard measurement protocols, establishment of emission reduction baselines, identification of mitigation techniques, and

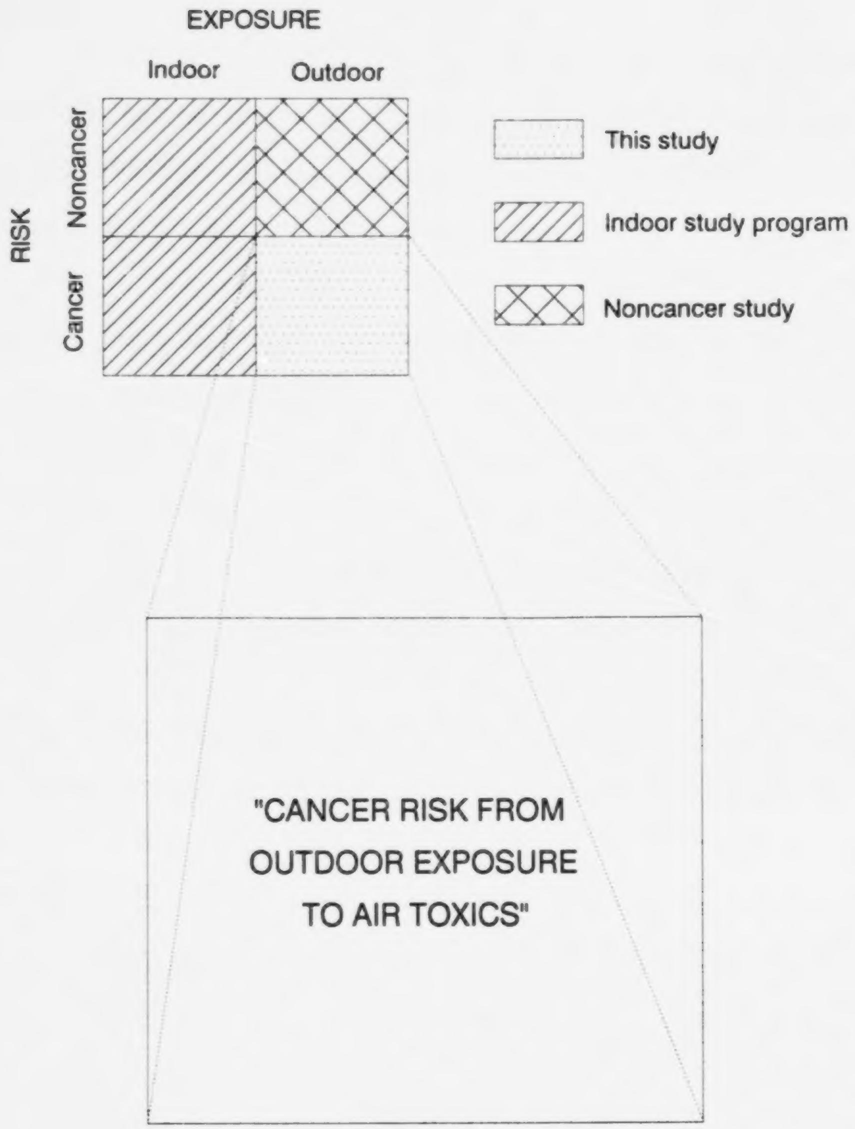
EXPOSURE
Indoor
Outdoor
RISK
Noncancer
Cancer
This study
Indoor study program
Noncancer study
"CANCER RISK FROM OUTDOOR EXPOSURE TO AIR TOXICS"

Figure 1-1. Relationship of this Study to Other Air Toxic Risk Studies

dissemination of information to the public. The EPA's ultimate goals in addressing indoor air quality problems are to characterize and understand the risks to human health that pollutants pose and to reduce those risks by reducing exposures. A report to Congress has been prepared that estimates cancer risk from indoor air toxics.[7]

Noncancer Health Risk Study[8]

In a separate study, EPA is evaluating the noncancer public health risks resulting from short-term and long-term outdoor exposure to toxic air pollutants. Noncancer effects range from subtle biochemical, physiological, or pathological effects to gross effects, including death. The main focus of the noncancer study is on the evaluation of risk from exposure to air toxics that are routinely emitted from industrial or commercial sources. Excluded from the noncancer analysis is the consideration of occupational exposures, indoor air pollutants, criteria air pollutants, secondary atmospheric reaction products, and accidental releases. The Executive Summary from the Noncancer Health Risk study is presented in Appendix C of this report.

SARA Title III

Under Title III of SARA, EPA is collecting air toxics emissions data from industrial and manufacturing sources that are covered by certain Standard Industrial Classification (SIC) codes, have 10 or more employees, and handle listed chemicals above threshold amounts. These data are collected as part of the Toxic Release Inventory mandated under

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[7] See Report to Congress on Indoor Air Quality (EPA-400/1-89-001, August 1989) for current estimates of cancer public health risks from exposure to indoor air toxics.

[8] U.S. Environmental Protection Agency, OAQPS. Toxic Air Pollutants and Noncancer Risks, Summary of Screening Study. External Review Draft, September, 1990.

Section 313 of SARA. Based on data contained in EPA's Toxic Release Inventory Data (1989), approximately 320 toxic chemicals were identified as being released to the environment and over 2.3 billion pounds of toxic chemicals were identified as being released to the air from the reporting facilities in 1987. Limiting the number of employees and specifying threshold amounts resulted in excluding smaller producers and facilities.

The following items highlight the relationship between the air toxic emissions data collected under SARA Title III and the risk estimates presented in this report.

- SARA Title III data concern only estimates of air toxics emissions and not estimates of cancer risk. This report focuses on estimates of cancer risk from exposure to air toxics. This report does not estimate emissions of air toxics, although the studies upon which the risk estimates are drawn had to estimate such emissions.
- SARA Title III emissions data are limited to industrial and manufacturing sources covered by SIC codes 20 through 39. This report is not limited to these sources, but includes additional emission sources such as mobile vehicles, treatment, storage, and disposal facilities for hazardous wastes (TSDFs), and dry cleaners.
- Generally, SARA Title III covers many more air toxics than this report, which focuses on the subset of pollutants for which cancer is the health concern and for which unit risk factors are available.
- The only information source used in this report that is similar to the SARA Title III effort is the Air Toxic Exposure and Risk Information System (ATERIS) data base, which includes nationwide emission estimates of many pollutants covered by SARA Title III.

The emission data submitted under SARA Title III were not used in this study to estimate cancer risk. The SARA Title III emission data are not reported in a form that allows estimation of risk. Thus, these data could not be used to estimate cancer risk for this study. However, the information on source emissions gathered under SARA Title III may be useful in identifying sources of concern for future risk assessments.

Outline of the Report

This report is divided into two volumes. Volume I contains a glossary of key terms; a list of acronyms; the Executive Summary; Chapter 1, Introduction; Chapter 2, Scope of Study and Analyses; Chapter 3, The Magnitude and Nature of the Cancer Risk; and Chapter 4, Summary and Conclusions. Volume II contains several appendices. The following paragraphs describe the remaining chapters of Volume I. This is then followed by a brief description of the material contained in Volume II.

In Chapter 2, "Scope of Study and Analyses," the various reports and information used, the analytical methodology used to develop estimates of annual cancer incidence, and major limitations and uncertainties associated with the risk estimates presented in the study are discussed.

The results of the study are presented in Chapter 3, "The Magnitude and Nature of the Cancer Risk." The magnitude of risk estimated, in terms of both estimated annual incidence and lifetime individual risk, is presented first. The nature of the cancer risk, in terms of individual pollutants, source categories, and geographic variability, is then presented. The results are then compared with those reported in the 1985 Six-Month Study.

Chapter 4, "Summary and Conclusions," summarizes the results of the study and presents the conclusions drawn from it with regard to the magnitude and nature of the cancer risk from outdoor air toxics.

As noted above, Volume II of this report contains the appendices. Appendix A lists the individuals who commented on the external review draft of this report and a summary of their comments. Appendix B provides detailed summaries of the analyses conducted for determining the estimates of cancer cases per year per million population that would be used in estimating total nationwide annual cancer incidence and the resulting estimates of total nationwide cancer incidence for each of those pollutants initially identified as possibly resulting in at least ten cancer cases per year nationwide. Appendix C provides summaries of the 14 EPA studies that focused on individual pollutants and source categories which formed part of the data base. As noted earlier, the Executive Summary to the Noncancer Health Risk study is also provided in Appendix C.

2.0 SCOPE OF STUDY AND ANALYSES

The purpose of this chapter is to provide the reader with an overview of the scope of the study, the analyses performed in estimating the cancer risk from air toxics, and an understanding of the limits and uncertainties associated with it. The scope is described by a discussion of the data base used. This discussion identifies for the reader the various reports and studies included and the pollutants and source categories examined. Next, the methodology used to derive the nationwide estimates of annual cancer incidence is described. This description gives the reader an understanding of the major components of the annual cancer incidence analysis, as well as some of its boundaries. Following the description of this analysis, limits and uncertainties associated with the risk estimates presented in this report are identified. By keeping in mind the scope of the study and the limits and uncertainties associated with these risk estimates, the reader will be able to more properly interpret and use the results of the study.

Data Base

A number of reports dealing with air toxics have been completed by EPA or other agencies since the 1985 Six-Month Study was published. A list of these reports was compiled and circulated to EPA Regional Offices, the State and Territorial Air Pollution Program Administrators and the Association of Local Air Pollution Control Officials (STAPPA/

ALAPCO), and others to identify any additional reports that might be included in this study. In addition to the reports, information from 14 individual source category- and pollutant-specific studies being conducted by EPA was included in this study. Two of these studies (the Superfund study and the Woodstove study) did not provide estimates of cancer risk that could be used to estimate nationwide cancer risk. Risk estimates based on the NESHAP (National Emission Standards for Hazardous Air Pollutants) analysis used for the 1985 Six-Month Study and supplemented by data contained in the Air Toxic Exposure and Risk Information System (ATERIS) data base developed by the EPA's Office of Air Quality Planning and Standards (OAQPS)[1] were also used in the analysis. As a result, the magnitude and nature of the cancer risk posed by air toxics were evaluated based upon information contained in ten reports, twelve source category- or pollutant-specific studies, and the NESHAP/ATERIS data base. The ten reports are listed in Table 2-1 and the fourteen source categories and pollutants for which information was obtained from other EPA studies are listed in Table 2-2.

The purposes of these reports and studies vary. Some were undertaken as general scoping studies to estimate cancer risk from air toxics in a specific locale (e.g., the Integrated Environmental Management Project (IEMP) studies, the South Coast Air Basin study) or on a national basis (e.g., the Mobile Source study, the Ambient Air Quality study). Some studies were undertaken to estimate cancer risk from a specific source category (e.g., publicly owned treatment works (POTWs), sewage sludge incinerators, mobile sources) or a specific pollutant

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[1] This is referred to in this study as the NESHAP/ATERIS data base. The NESHAP risk estimates from the 1985 Six-Month Study were updated by applying new unit risk factors for those pollutants whose unit risk factors had changed since the original analysis.

TABLE 2-1

LIST OF REPORTS USED IN STUDY

1. U.S. EPA, Region III. Kanawha Valley Toxics Screening Study, Final Report. July 1987. (IEMP-Kanawha Valley)

2. U.S. EPA, OPPE. Santa Clara Valley Integrated Environmental Management Project: Revised Stage I Report. May 30, 1986. (IEMP-Santa Clara)

3. U.S. EPA, OPPE. Baltimore Integrated Environmental Management Project: Phase I Report. May 1987. (IEMP-Baltimore)

4. U.S. EPA, Region V. Estimation and Evaluation of Cancer Risk Attributable to Air Pollution in Southeast Chicago (Draft). January 1989. (Southeast Chicago)

5. U.S. EPA, OAQPS. Analysis of Air Toxics Emissions, Exposures, Cancer Risks and Controllability in Five Urban Areas. Volume I, Base Year Analysis and Results. EPA-450/2-89-012a. July 1989. (5 City)

6. U.S. EPA, OAQPS. Updated Estimated Cancer Incidence for Selected Toxic Air Pollutants Based on Ambient Air Pollution Data. August 1989. (Ambient Air Quality)[a]

7. South Coast Air Quality Management District. The Magnitude of Ambient Air Toxics Impacts from Existing Sources in the South Coast Air Basin. 1987 Air Quality Management Plan Revision Working Paper No. 3. June 1987. (South Coast)[b]

8. U.S. EPA, OPPE. Final Report of the Philadelphia Integrated Environmental Management Project. December 1986. (IEMP-Philadelphia)

9. American Management Systems. Updated 35-County Study. March 1988. (35-County) This report was prepared under contract to the U.S. EPA.

10. U.S. EPA, Office of Mobile Sources. Air Toxics Emissions from Motor Vehicles. EPA-AA-TSS-PA-86-5. (Mobile Sources)[c]

FOOTNOTES TO TABLE 2-1

[a] The VOC data used in this study was obtained from either: (1) J.J. Shah and E. K. Heyerdahl, National Ambient Volatile Organic Compounds (VOC's) Data Base Update, U.S. EPA, Atmospheric Sciences Research Laboratory, Research Triangle Park, NC, February 1988, or (2) A. Pollack, Systems Applications, Inc., Updated Report on the Interim Data Base for State and Local Air Toxic Volatile Organic Chemical Measurements, prepared for Bob Faoro, U.S. EPA, OAQPS, Research Triangle Park, NC, August 1988. The trace metal data were obtained from the Aerometric Information Retrieval System, U.S. EPA, OAQPS, Research Triangle Park, NC, March 1988, and the benzo(a)pyrene (BaP) data from J. Bumgarner, Environmental Monitoring and Systems Laboratory, U.S. EPA, Research Triangle Park, NC, September 1988.

[b] Reprinted by the U.S. Environmental Protection Agency as Multiple Air Toxics Exposure Study, Working Paper No. 3, South Coast Air Basin, EPA-450/4-88-013, November 1988.

[c] Information in this study has been updated in this report using "Air Toxics Emissions from Motor Vehicles," prepared by Penny Carey and Joseph Somers. This paper was presented at the 81st Meeting of APCA, Dallas, Texas, June 19-24, 1988.

TABLE 2-2

EPA SOURCE CATEGORY AND POLLUTANT STUDIES

Source Categories

1. Coal and oil combustion
2. Drinking water aerators
3. Gasoline marketing
4. Hazardous waste combustors
5. Municipal incinerators
6. Municipal solid waste landfills
7. Publicly owned treatment works (POTWs)
8. Sewage sludge incinerators
9. Superfund sites
10. Treatment, storage, and disposal facilities for hazardous waste (TSDFs)
11. Waste oil combustors
12. Woodstoves

Pollutants

13. Asbestos
14. Radionuclides

NOTE: The references used to obtain risk estimates from these source category and pollutant studies are identified in Appendix C.

(e.g., radionuclides, asbestos). The ATERIS data base contains information generated from assessments of potentially toxic air pollutants performed by OAQPS. The ATERIS contains data from all stages of air toxics analyses, from the very preliminary to the more detailed analyses. The data contained in the ATERIS are intended for the relative comparison and ranking of source categories and pollutants on a nationwide basis. The information in ATERIS is not considered an authoritative source for verified estimates of risk attributable to individual point sources.

The number of pollutants and source categories included in the individual studies varied. As shown in Table 2-3, the number of pollutants contributing to the estimated cancer risk in a study varied from one (the Asbestos study) to 74 (the Hazardous Waste Combustor study). The study for the treatment, storage, and disposal facilities for hazardous waste (TSDFs) used an initial list of 84 potential air pollutants, 74 of which were identified as being emitted. Of these 74 pollutants, risk estimates for 42 were made on the basis of available EPA unit risk factors. Most studies included 9 to 20 individual pollutants in their risk estimates.

A total of 90 different pollutants[2] were included in the 22 studies and reports (see Table 2-4). Forty-eight of the pollutants were included in one or two studies. Most of these 48 pollutants were found in the NESHAP/ATERIS data base, the Hazardous Waste Combustor study, the Sewage Sludge Incinerator study, or the TSDF study. Another 22 pollutants were found in three to six studies. Twenty pollutants were included in more than six studies.

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[2] Not all of these pollutants, however, have EPA-derived unit risk factors, as shown in Tables 2-6 and 2-7 and as indicated in Table 2-4.

TABLE 2-3

NUMBER OF POLLUTANTS INCLUDED IN CANCER INCIDENCE ESTIMATES, BY STUDY

	STUDY	NUMBER OF POLLUTANTS INCLUDED IN RISK ESTIMATE
1.	NESHAP/ATERIS Data Base	45
2.	Ambient Air Quality	20
3.	35-County	19
4.	5-City	19
5.	IEMP-Baltimore	9
6.	IEMP-Kanawha Valley	18
7.	IEMP-Philadelphia	9
8.	IEMP-Santa Clara	14
9.	South Coast	15
10.	Southeast Chicago	30
11.	Mobile Sources	9
12.	Asbestos	1
13.	Coal and Oil Combustion	9
14.	Drinking Water Aerators	10
15.	Gasoline Marketing	4
16.	Hazardous Waste Combustors	74
17.	Municipal Waste Combustors	10
18.	POTWs	7
19.	Radionuclides	2
20.	Sewage Sludge Incinerators	33
21.	TSDFs	42
22.	Waste Oil Combustors	8

NOTE: The Municipal Solid Waste Landfills, Superfund Sites, and Woodstove studies do not include estimates of cancer incidence.

TABLE 2-4

NUMBER OF STUDIES THAT INCLUDED SPECIFIC POLLUTANT IN CANCER RISK ESTIMATE, BY POLLUTANT

POLLUTANT	NUMBER OF STUDIES
1. Acetaldehyde	3
2. Acrolein[a]	1
3. Acrylamide	4
4. Acrylonitrile	8
5. Aldrin	3
6. Allyl chloride	3
7. Aniline	2
8. Arsenic	13
9. Asbestos	4
10. Benz(a)anthracene	2
11. Benzene	17
12. Benzidine	2
13. Benzo(a)pyrene (BaP)	6
14. Benzyl chloride[a]	2
15. Beryllium	12
16. Bis(2-chloroethyl) ether	1
17. Bis(chloromethyl) ether	2
18. Bis(2-ethyhexyl) phthalate	1
19. 1,3-Butadiene	9
20. Cadmium	15
21. Carbon tetrachloride	15
22. Chlordane	3
23. Chloroform	15
24. Chloromethane	2
25. Chlorophenols[b]	4
26. Chromium (VI)	13
27. Coke Oven Emissions	3
28. DDT	2
29. Dibenz(a,h)anthracene	3
30. 1,2-Dibromo-3-chloropropane	4
31. p-dichlorobenzene	1
32. 1,2-Dichloropropane	3
33. Dieldrin	2
34. Diethylstilbestrol	2
35. Diethanolamine[a]	1
36. Dimethylnitrosamine	1
37. 2,4-Dinitrotoluene	2
38. Dioctyl phthalate[a]	1
39. 1,4-Dioxane	2
40. Dioxin	6
41. 1,2-Diphenyl hydrazine	2
42. Epichlorohydrin	4
43. Ethyl acrylate[a]	2
44. Ethylene dibromide[c]	12
45. Ethylene dichloride[d]	14
46. Ethylene oxide	7
47. Formaldehyde	11
48. Gasoline vapors	8
49. Heptachlor	2
50. Heptachlor epoxide	2
51. Hexachlorobenzene	4
52. Hexachlorobutadiene	2
53. gamma-Hexachlorocyclohexane (lindane)	2
54. Hexachloroethane	2
55. Hydrazine/Hydrazine sulfate	2
56. 4,4 Isopropylidene diphenol[a]	1
57. Methyl chloride	3
58. 3-Methylchloanthrene	3
59. Methyl hydrazine	1
60. Methylene chloride	13
61. 4,4-Methylene dianiline[a]	1
62. Nickel (subsulfide)	6
63. Nitrobenzene[a]	2
64. 2-Nitropropane	3
65. n-Nitroso-n-butylamine	1
66. n-Nitro-n-methylurea	1
67. n-Nitrosodiethylamine	2
68. Nitrosomorpholine[a]	1
69. N-Nitrosopyrrolidine	1
70. Pentachloronitrobenzene	1
71. Perchloroethylene	16
72. PIC[e]	8
73. PCB[f]	7
74. Pronamide	1
75. Propylene dichloride[a]	2
76. Propylene oxide	2
77. Radionuclides	2
78. Radon	1
79. Reserpine	2
80. Styrene	4
81. Terephthalic acid[a]	1

TABLE 2-4 (concluded)

NUMBER OF STUDIES THAT INCLUDED SPECIFIC POLLUTANT IN CANCER RISK ESTIMATE, BY POLLUTANT

POLLUTANT	NUMBER OF STUDIES	POLLUTANT	NUMBER OF STUDIES
82. 1,1,2,2-Tetrachloroethane	3	86. 1,1,1-Trichloroethane[a]	1
83. Thiourea	2	87. Trichloroethane	2
84. Titanium dioxide[a]	1	88. Trichloroethylene	16
85. Toxaphene	2	89. Vinyl chloride	11
		90. Vinylidene chloride	6

[a] EPA-derived unit risk factors not available.

[b] Includes pentachlorophenol and trichlorophenol. Only an EPA unit risk factor for trichlorophenol is available.

[c] 1,2-dibromoethane.

[d] 1,2-dichloroethane.

[e] PIC = products of incomplete combustion.

[f] PCB = polychlorinated biphenyls.

As shown in Table 2-5, 65 source categories were identified from among the studies and reports. Two of the source categories are general in their coverage. These are: (1) chemical manufacturing (unspecified) and (2) unspecified sources. Forty-five of the source categories were identified as being in only one study. Most of these source categories were identified in the NESHAP/ATERIS data base. Nine of the source categories were included in four or more studies, with gasoline marketing included in the most (nine) studies. It is likely that some of the specified source categories are included in the "chemical manufacturing (unspecified)" and the "unspecified" categories.

Annual Cancer Incidence Analysis

The total nationwide estimate of cancer incidence was based on the estimated cancer incidence from all pollutants for which unit risk factors have been developed by EPA and from all source categories covered by the studies and reports in the data base. It is important to understand that not all of the unit risk factors developed by EPA have undergone the same level of scrutiny. In general, many of the unit risk factors (e.g., those for benzene and carbon tetrachloride) have been "verified" by the Agency, having undergone review by an Agency work group, the Carcinogen Risk Assessment Verification Endeavor. Such unit risk factors are identified in Table 2-6 by reference to the Integrated Risk Information System. Most of the unit risk factors, however, have not been Agency-verified. The non-verified unit risk factors have undergone various levels of review. Some have received review by the Office of Health and Environmental Assessment. Others have received little review. Among the least reviewed unit risk factors are those estimated for the group of compounds referred to in this study as

TABLE 2-5

DISTRIBUTION OF SOURCE CATEGORIES BY NUMBER OF STUDIES

SOURCE CATEGORY	NUMBER OF STUDIES	SOURCE CATEGORY	NUMBER OF STUDIES
1. ABS/SAN production[a]	1		
2. Acrylic fiber production	1	34. Gasoline Marketing	9
3. Acrylonitrile monomer	1	35. Glass mfg.	1
4. Asbestos, demolition	1	36. Hexamethylenetetram mfg.	1
5. Asbestos, fabrication	1	37. Ind. solvent coatings	1
6. Asbestos, manufacturing	1	38. Iron and Steel mfg.	4
7. Asbestos, milling	1	39. Melamine formaldehyde resin	1
8. Asbestos, renovation	1	40. 4,4-Methylenedianiline	1
9. Benzene fugitives	1	41. Motor vehicles	8
10. Benzene storage	1	42. Municipal solid waste landfills	2
11. Benzene usage	1	43. Municipal waste combustors	4
12. 1,3-Butadiene production	1	44. Nitrile elastomer evaporation	1
13. 1,4-Butanediol	1	45. Other organic evaporation	1
14. Cadmium pigment mfg.	1	46. Pentaerythitol production	1
15. Cadmium stabilizer mfg.	1	47. Pesticide Production/ Usage	2
16. Carbon tetrachloride production	1	48. Petroleum Refineries	2
17. Chemical manufacturing (unspecified)	6	49. Pharmaceutical mfg.	1
18. Chlorinated drinking water	1	50. Phenol formaldehyde resins	1
19. Chlorine production	1	51. Phthalic anhydride	1
20. Chlorinated hydrocarbon production	1	52. Polyacetal resins	1
21. Chloroflourocarbon production	1	53. Polybutadiene production	1
22. Chlorinated hydrocarbon	1	54. Publicly owned treatment works	6
23. Chlorinated hydrocarbon users	1	55. Pulp and paper mfg.	1
24. Coal and Oil Combustion/ Heating	5	56. Sewage sludge incinerators	2
25. Commercial Sterilization/ Hospitals	2	57. Solvent Use/Degreasing	5
26. Cooling Towers	3	58. SBR production[e]	1
27. Drinking Water Aerators	2	59. Stripping (paint, photo-resist)	1
28. Drycleaning	4	60. TSDFs[f]	2
29. EBS production[b]	1	61. Trimethylopropane	1
30. EDB manufacturing[c]	1	62. Unspecified	7
31. Electroplating	4	63. Urea Formaldehyde	1
32. ETO production[d]	1	64. Waste Oil Burning	3
33. Formaldehyde production	1	65. Woodsmoke	1

FOOTNOTES TO TABLE 2-5

[a] ABS/SAN = acrylonitrile butadiene styrene/styrene acrylonitrile

[b] EBS = ethylbenzene styrene

[c] EDB = ethylene dibromide

[d] ETO = ethylene oxide

[e] SBR = styrene butadiene rubber

[f] TSDF = treatment, storage, and disposal facilities for hazardous waste

products of incomplete combustion (PIC).[3] Finally, many of the unit risk factors remain uncertain and are subject to change as further evidence of carcinogenicity is obtained.

Primarily due to the limited time and resources available for the report, the annual cancer incidence analysis was limited in two aspects:

- The analysis did not try to verify the results of the various studies. Any errors that might be contained in the studies would, therefore, be carried over into this study. In a few instances, some information was double-checked as calculations suggested a possible error or two. Double checking of information was the exception, however, and not the rule.
- The initial analysis was carried out on the basis of readily available background documents and reports. In some instances, the documents and reports did not provide all of the necessary level of detail that would have been preferred. This left a level of uncertainty in trying to compare data and resolve differences. In general, these instances have been identified in the pollutant-by-pollutant analysis summaries, which are found in Appendix B.

Methodology

The annual cancer incidence analysis began by assembling the annual cancer incidence estimates for each pollutant by source category from each of the 22 studies. Because the 22 studies varied in geographic scope and thus population exposed, the annual cancer incidence estimates were of limited value by themselves, especially where the study was of limited geographic scope. Therefore, an attempt was made to correct for geographic scope by calculating the cancer incidence per year per million population for each pollutant in each source category for each study. For the smaller, localized studies, the population reported in the studies was used to calculate the annual cancer incidence per million population. For each nationwide study, a 1986 population of 240 million was used rather than trying to determine the base year for each nationwide study.[4]

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[3] In this study, PIC refers to the large number of primarily particulate compounds that result from incomplete combustion. PIC is composed primarily of "polycyclic organic matter" (POM). Some studies use the term POM when estimating the risk from this class of compounds. In addition, some studies use benzo(a)pyrene (BaP), which is a component of POM, as a surrogate to estimate risk from PIC.

The various pollutants and source categories frequently "overlapped" between reports; that is, the same pollutant/source category combination (e.g., 1,3-butadiene emissions from motor vehicles) was included in more than one study or report. Figure 2-1 illustrates this overlap in a simplified diagram for five hypothetical studies. Studies No. 1 and 2 represent some of the larger studies, such as the 35-County study or the 5-City study. Study No. 3 represents a source category specific study. Studies No. 4 and 5 represent pollutant specific studies. For example, Study No. 2 is seen in Figure 2-1 to cover three of the same pollutants for two source categories as Study No. 1, and the same three pollutants for one source category as Study No. 3. Study No. 5 overlaps one pollutant/source category combination with Study No. 3. Study No. 4 covers some of the same source categories found in Studies No. 1 and 2, but for a different pollutant.

Where overlaps of pollutants and source categories occurred, the estimates of annual cancer incidences per million population from each study were compared. If the estimates were the same (or essentially the same) for a pollutant/source category across all studies, additional analysis to identify potential causes for differences was obviously

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[4] The risk estimates in all of the studies and reports used in this study are based on 1980 to 1987 data (i.e., emission inventories, ambient measurements, populations, etc.). For purposes of this study, these data were treated as applying to the same time frame. The risk estimates can be considered as mid-1980 numbers, or 1986 estimates.

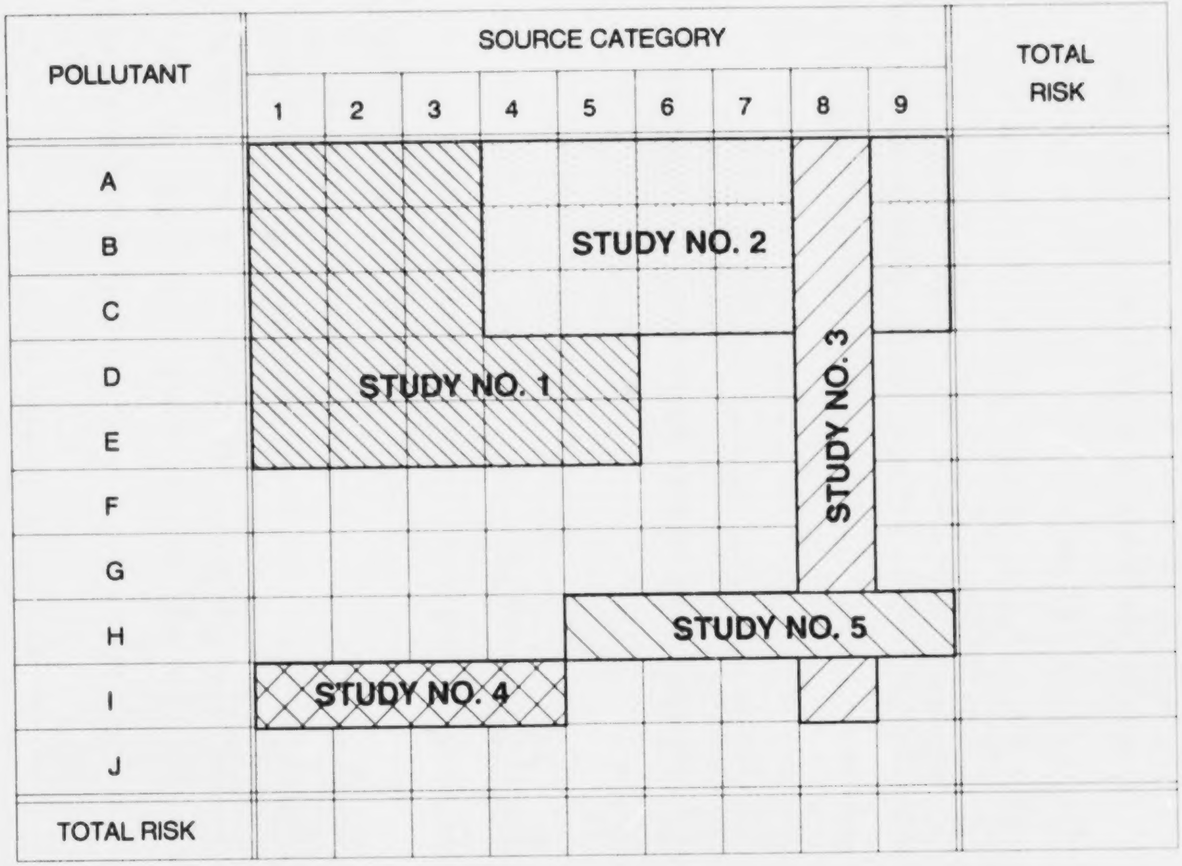
POLLUTANT
SOURCE CATEGORY
1
2
3
4
5
6
7
8
9
TOTAL RISK
A
B
C
D
E
F
G
H
I
J
TOTAL RISK
STUDY NO. 1
STUDY NO. 2
STUDY NO. 3
STUDY NO. 4
STUDY NO. 5

Figure 2-1. Illustrative Relationship of Pollutants and Source Categories Covered by Five Hypothetical Studies

unnecessary. If differences in the estimates of annual cancer incidence per million population were found within a pollutant/source category combination, a reduction analysis[5] (as discussed below) was conducted to resolve the differences and develop a point estimate[6] of the annual cancer incidence per million population for that pollutant/source category combination. If a pollutant/source category combination was unique to an individual study, the estimate of annual cancer incidence per million population for that pollutant/source category was considered the best available estimate. Once the point estimates of annual cancer incidence per million population were identified, they were adjusted, as necessary, to common unit risk factors for each pollutant. (This adjustment is discussed later in this chapter under the Reduction Analysis section.)

In extrapolating the estimates of annual cancer incidence per million population to total nationwide annual incidence, the geographic scope of the study was considered. Most pollutants and source categories were in at least one study that was nationwide in scope. This enabled, in most instances, a direct extrapolation to total nationwide estimates (i.e., multiplying the cancer rate by the total U.S. population of 240 million). A few pollutants and source categories were included only in a study of limited geographic scope. In such instances, an attempt was made to determine whether the pollutant/source category was unique to the geographic area, unusually concentrated in the area, or fairly common across the United States. If it was unique to the area or appeared to be unusually concentrated in the area, then generally only the cancer incidence estimated in the study for that category was included in the total nationwide estimate. If the pollutant/source category appeared to be fairly widespread, the estimate of annual cancer incidence per million population was extrapolated to a total nationwide estimate (i.e., multiplied by 240 million population).

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[5] This type of reduction analysis was not undertaken for individual risks because individual risks are site-specific numbers that cannot be extrapolated to a nationwide estimate of individual risk. Instead, the study presents the estimates of individual risk as found in each of the studies used in the data base for this study.

[6] In some instances, it was not possible to develop a point estimate. In such cases, the range of estimates for the cancer rate was narrowed as much as possible.

Once this was done, the estimates of risk for each pollutant/source category combination were summed to calculate the nationwide estimate of annual cancer incidence.

Reduction Analysis. As noted previously, a large number of pollutant/source category combinations with discrepant estimates of annual cancer incidence per million population were identified. An analysis was undertaken in an attempt to derive a single estimate of the annual cancer incidence per million population.

A decision was made to limit the number of pollutant/source category combinations for which the reduction analysis would be conducted. It was decided to analyze the estimates of annual cancer incidence per million population of pollutant/source category combinations for those pollutants that could potentially result in 10 or more cancer cases per year nationwide based on information in any one study. These pollutants were identified in one of two ways:

(1) by the total number of annual cancer cases estimated for them in studies that were nationwide in scope (e.g., the Ambient Air Quality study, the Mobile Source study); or

(2) by the calculated number of cancer cases per year per million population which when extrapolated nationwide might result in 10 or more cancer cases per year for the smaller geographic studies (e.g., the four IEMP studies).

A total of 23 pollutants were identified. It is these 23 pollutants that are presented in Appendix B.

The reduction analysis looked to identify and reduce the discrepancies by analyzing the following set of factors:

- unit risk factors
- emission factors
- modeled vs. ambient-measured concentrations
- source category definition and coverage
- geographic scope of the study
- study specific considerations

Each of these factors are discussed below as to how they were used and considered in the reduction analysis.

Unit Risk Factors.[7] Perhaps the most obvious reason that two estimates of annual cancer incidence per million population would differ is that a different unit risk factor had been used. Unit risk factors have changed in the past and may change in the future. Thus, the first step in the analysis was to put these estimates on the same "footing"; that is, making sure the risk estimates are compared using the same unit risk factors. The unit risk factors used in each study[8] were compared to those identified in Table 2-6 and Table 2-7. Table 2-7 shows the unit risk factors used to estimate the cancer risk from PIC. The pollutants

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[7] The unit risk factor is a quantitative estimate of the carcinogenicity potency of a pollutant. It is often expressed as the chance of contracting cancer from a 70-year lifetime continuous exposure to a concentration of one microgram per cubic meter (1 $\mu g/m^3$) of a given pollutant. For example, benzene has a unit risk factor of $8.3x10^{-6}$ $(\mu g/m^3)^{-1}$. In a population of 100,000 people exposed to 10 $\mu g/m^3$ of benzene for 70 years, the upper-bound estimate of cancer cases is calculated to be 8.3 cancer cases over 70 years (10 $\mu g/m^3$ x 100,000 people x $8.3x10^{-6}$ $(\mu g/m^3)^{-1}$ = 8.3 cancer cases over 70 years).

[8] Unit risk factors used in the Municipal Waste Combustor study were not in the available reports, and were assumed to be the same as those in Table 2-6. Unit factors for radionuclides and radon were accepted "as is" in the reports.

TABLE 2-6

UNIT RISK FACTORS USED TO COMPARE CANCER RISK

POLLUTANT (CAS NO.)	EPA CLASSIFICATION[a]	UNIT RISK FACTORS $(\mu g/m^3)^{-1}$	REFERENCE
1. Acetaldehyde (75-07-0)	B2	2.2×10^{-6}	1
2. Acrylamide (79-06-1)	B2	1.1×10^{-3}	2
3. Acrylonitrile (107-13-1)	B1	6.8×10^{-5}	1
4. Aldrin (309-00-2)	B2	4.9×10^{-3}	1
5. Allyl chloride (107-05-1)	B2	5.5×10^{-8}	3
6. Aniline (62-53-3)	B2	7.4×10^{-6}	2
7. Arsenic (7440-38-2)	A	4.3×10^{-3}	1
8. Asbestos (1332-21-4)	A	7.6×10^{-3}	1[b]
9. Benz(a)anthracene (56-55-3)	B2	8.9×10^{-4}	2
10. Benzene (71-43-2)	A	8.3×10^{-6}	1
11. Benzidine (92-87-5)	A	6.7×10^{-2}	1
12. Beryllium (7440-41-7)	B2	2.4×10^{-3}	1
13. Bis(2-chloroethyl)ether (111-44-4)	B2	3.3×10^{-4}	1
14. Bis(chloromethyl)ether (542-88-1)	A	2.7×10^{-3}	2[c]
15. Bis(2-ethylhexyl)phthalate (117-81-7)	B2	2.4×10^{-7}	4
16. 1,3-Butadiene (106-99-0)	B2	2.8×10^{-4}	1
17. Cadmium (7440-43-9)	B1	1.8×10^{-3}	1
18. Carbon tetrachloride (56-23-5)	B2	1.5×10^{-5}	1
19. Chlordane (12789-03-6)	B2	3.7×10^{-4}	1
20. Chloroform (67-66-3)	B2	2.3×10^{-5}	1
21. Chloromethane (74-87-3)	--	3.6×10^{-6}	4
22. Chromium (VI) (7440-47-3)	A	1.2×10^{-2}	1
23. Coke Oven Emissions	A	6.2×10^{-4}	1
24. DDT (50-29-3)	B2	3.0×10^{-4}	2
25. Dibenz(a,h)anthracene (53-70-3)	B2	1.4×10^{-2}	2
26. 1,2-Dibromo-3-chloropropane (96-12-8)	B2	6.3×10^{-3}	2
27. 1,2-Dichloropropane (78-87-5)	C	1.8×10^{-5}	5
28. Dieldrin (60-57-1)	B2	4.6×10^{-3}	2
29. Diethylstilbesterol (56-53-1)	--	1.4×10^{-1}	4
30. Dimethylnitrosamine (62-75-9)		1.4×10^{-2}	1
31. 2,4-Dinitrotoluene (121-14-2)	B2	8.8×10^{-5}	2
32. 1,4-Dioxane (123-91-1)	B2	1.4×10^{-6}	2
33. Dioxin (1746-01-6)	B2	3.3×10^{1}	2

TABLE 2-6 (continued)

UNIT RISK FACTORS USED TO COMPARE CANCER RISK

POLLUTANT (CAS NO.)	EPA CLASSIFICATION[a]	UNIT RISK FACTORS $(\mu g/m^3)^{-1}$	REFERENCE
34. 1,2-Diphenylhydrazine (122-66-7)	B2	$2.2x10^{-4}$	1
35. Epichlorohydrin (106-89-8)	B2	$1.2x10^{-6}$	1
36. Ethylene dibromide (106-93-4)	B2	$2.2x10^{-4}$	1
37. Ethylene dichloride (107-06-2)	B2	$2.6x10^{-5}$	1
38. Ethylene oxide (75-21-8)	B1-B2	$1.0x10^{-4}$	2
39. Formaldehyde (50-00-0)	B1	$1.3x10^{-5}$	1
40. Gasoline vapors (8006-61-9)	B2	$6.6x10^{-7}$	2
41. Heptachlor (76-44-8)	B2	$1.3x10^{-3}$	1
42. Heptachlor epoxide (1024-57-3)	B2	$2.6x10^{-3}$	1
43. Hexachlorobenzene (118-74-1)	B2	$4.9x10^{-4}$	4
44. Hexachlorobutadiene (87-68-3)	C	$2.2x10^{-5}$	1
45. gamma-Hexachlorocyclohexane (lindane) (58-89-9)	C	$3.8x10^{-4}$	2
46. Hexachloroethane (67-72-1)	C	$4.0x10^{-6}$	1
47. Hydrazine (302-01-2)	B2	$2.9x10^{-3}$	4[d]
48. Methyl chloride (74-87-3)	C	$3.6x10^{-6}$	2
49. 3-Methylchloanthrene (56-49-5)	B2	$2.7X10^{-3}$	2
50. Methyl hydrazine (60-34-4)	B2	$3.1x10^{-4}$	2
51. Methylene chloride (75-09-2)	B2	$4.7x10^{-7}$	2
52. Nickel (subsulfide) (12035-72-2)	A	$4.8x10^{-4}$	1
53. 2-Nitropropane (79-46-9)	B2	$2.7x10^{-3}$	2
54. n-Nitrosodi-n-butylamine (924-16-3)	B2	$1.6x10^{-3}$	1
55. n-Nitrosodiethylamine (55-18-5)	B2	$4.3x10^{-2}$	2
56. n-Nitroso-n-methylurea (684-93-5)	B2	$8.6x10^{-2}$	2
57. n-Nitrosopyrrolidine (930-55-2)	B2	$6.1x10^{-4}$	1
58. Pentachloronitrobenzene (82-68-8)	C	$7.3x10^{-5}$	2
59. Perchloroethylene (127-18-4)	B2	$5.8x10^{-7}$	2

TABLE 2-6 (concluded)

UNIT RISK FACTORS USED TO COMPARE CANCER RISK

POLLUTANT (CAS NO.)	EPA CLASSIFICATION[a]	UNIT RISK FACTORS $(\mu g/m^3)^{-1}$	REFERENCE
60. PCB's (1336-36-3)	B2	$1.2x10^{-3}$	2
61. Pronamide (23950-58-5)	C	$4.6x10^{-6}$	2
62. Propylene oxide (75-56-9)	B2	$3.7x10^{-6}$	6
63. Reserpine (50-55-5)	B2	$3.0x10^{-3}$	2
64. Styrene (100-42-5)	B2	$5.7x10^{-7}$	6
65. 1,1,2,2-Tetrachloroethane (79-34-5)	C	$5.8x10^{-5}$	1
66. Thiourea (62-56-6)	B2	$5.5x10^{-4}$	2
67. Toxaphene (8001-35-2)	B2	$3.2x10^{-3}$	1
68. 1,1,2-Trichloroethane (79-00-5)	C	$1.6x10^{-5}$	1
69. Trichloroethylene (79-01-6)	B2	$1.7x10^{-6}$	2
70. 2,4,6-Trichlorophenol (88-06-2)	B2	$5.7x10^{-6}$	2[e]
71. Vinyl chloride (75-01-4)	A	$4.1x10^{-6}$	7[f]
72. Vinylidene chloride (75-35-4)	C	$5.0x10^{-5}$	1

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[a] For a discussion of how EPA evaluates suspect carcinogens and more information on these classifications, refer to "Guidelines for Carcinogen Risk Assessment" (51 Federal Register 33992). The EPA classifications used in this report are:

A = proven human carcinogen

B = probable human carcinogen (B1 indicates limited evidence from human studies and sufficient evidence from animal studies; B2 indicates sufficient evidence from animal studies but inadequate evidence from human studies)

C = possible human carcinogen

[b] Derived from 2.3×10^{-1} per fibers per ml (millimeter), which is the unit risk factor reported in IRIS (Integrated Risk Information System)

[c] IRIS currently reports a unit risk factor of $6.2x10^{-2}$ $(\mu g/m^3)^{-1}$.

[d] IRIS currently reports a unit risk factor of $4.9x10^{-3}$ $(\mu g/m^3)^{-1}$.

[e] IRIS currently reports a unit risk factor of $3.1x10^{-6}$ $(\mu g/m^3)^{-1}$.

[f] An alternative unit risk factor of $4.2x10^{-5}$ $(\mu g/m^3)^{-1}$ has been developed by ORD. U.S. Environmental Protection Agency, Office of Research and Development, Office of Health and Environmental Assessment. Health Effects Assessment Summary Tables First Quarter FY89. January 1989.

REFERENCES TO TABLE 2-6

1. U.S. EPA, Office of Health and Environmental Assessment. Cincinnati, Ohio. Integrated Risk Information System (on-line data base).

2. U.S. Environmental Protection Agency. Hazardous Waste TSDF - Background Information for Proposed RCRA Air Emission Standards, Volume II - Appendices. Preliminary Draft. March 1988. pp. E-8 through E-13.

3. Schell, R.M. Estimation of the Public Health Risks Associated with Exposure to Ambient Concentrations of 87 Substances. OAQPS, U.S. EPA, July, 1984. Revised February 1985.

4. U.S. EPA, Office of Solid Waste. Draft Supplemental Rule for Hazardous Waste Incinerators. Appendix B, Unit Risks for Carcinogenic Constituents. January 16, 1989.

5. IEMP-Philadelphia. Developed from EPA's Drinking Water Criteria Document. March 2, 1984.

6. U.S. Environmental Protection Agency, Office of Research and Development, Office of Health and Environmental Assessment. Health Effects Assessment Summary Tables Third Quarter FY90. July 1990.

7. U.S. Environmental Protection Agency, Office of Research and Development, Office of Health and Environmental Assessment. Health Effects Assessment Summary Tables First Quarter FY89. January 1989.

TABLE 2-7

UNIT RISK FACTORS USED TO ESTIMATE CANCER RISK FROM PIC

SOURCE CATEGORY	COMPONENT	UNIT RISK FACTOR $(\mu g/m^3)^{-1}$	REFERENCE
Unspecified	BaP	1.7×10^{-3}[a]	1
Unspecified[b]	PIC	4.2×10^{-1}	2
Coke Ovens[c]	POM	6.5×10^{-5}	3
Municipal Incinerators[c]	POM	8×10^{-8}	3
Industrial power plants, oil[c]	POM	3.0×10^{-7}	3
Utility power plants, oil[c]	POM	3.0×10^{-7}	3
Industrial power plants, coal[c]	POM	8×10^{-8}	3
Utility power plants, coal[c]	POM	8×10^{-8}	3
Residential Heating[c]			3
Oil	POM	9×10^{-6}	
Coal	POM	1.0×10^{-5}	
Wood	POM	1.0×10^{-5}	
Gasoline vehicles	POM	2.5×10^{-4}	4
Diesel vehicles	POM	2.0×10^{-5} to 10×10^{-5}	4
Sewage Sludge Incinerators	BaP	3.7×10^{-6}	5
Hazardous Waste Combustors	PIC	1.0×10^{-5}	5

[a] Based on inhalation study. Oral study suggests a unit risk factor of 3.3×10^{-3} $(\mu g/m^3)^{-1}$.

[b] This unit risk factor for products of incomplete combustion (PIC) was based on relating lung cancer deaths to benzo(a)pyrene (BaP) concentrations where BaP serves as a surrogate for the large category of BaP-related pollutants referred to in the Six-Month Study as PIC. For a more detailed explanation of its derivation, refer to pages 20 to 24a of the Six-Month Study.

[c] These factors have been adjusted such that they are applied to the total particulate concentration to estimate risk from the POM fraction of the particulate matter.

REFERENCES TO TABLE 2-7

1. U.S. Environmental Protection Agency. Hazardous Waste TSDF - Background Information for Proposed RCRA Air Emission Standards, Volume II - Appendices. Preliminary Draft. March 1988. pp. E-8 through E-13.

2. U.S. EPA, Office of Policy, Planning, and Evaluation. The Air Toxics Problem in the United States: An Analysis of Cancer Risks for Selected Pollutants. EPA-450/1-85-001. May 1985.

3. U.S. EPA. Analysis of Air Toxics Emissions, Exposures, Cancer Risks and Controllability in Five Urban Areas. EPA-450/2-89-012a. July 1989.

4. U.S. EPA, Office of Mobile Sources. Air Toxics Emissions From Motor Vehicles, September 1987.

5. Memorandum. Shiva Garg, US EPA, Office of Solid Waste and Emergency Response, to Joseph Padgett, US EPA, Office of Air Quality Planning and Standards. Review of OAQPS Report on Six-Month Study of Impacts of Air Toxics on Cancer Incidence. March 3, 1989.

in each study that had different unit risk values than those shown in Table 2-6 are identified in Table 2-8.

The estimates of cancer incidence for PIC reported in Chapter 3 and the Executive Summary are based primarily on the unit risk factors specific to individual source categories that are shown in Table 2-7. The unit risk factor for PIC of 4.2×10^{-1} $(\mu g/m^3)^{-1}$ for unspecified sources was used only if a source-specific PIC unit risk factor was not available. The method used to calculate this PIC unit risk factor was unusual, and any risk estimate based on its use should be treated as a very preliminary estimate. Some of the studies, such as the Ambient Air Quality study, used this unit risk factor to estimate risk from PIC using benzo(a)pyrene (BaP) ambient-measured concentrations as a surrogate for PIC exposure. Some studies also used this unit risk factor for purposes of comparing cancer incidence estimates using various methodologies. For a discussion of these methodologies, please refer to the section on PIC found in Appendix B.

If a pollutant's unit risk factor differed from that in Table 2-6, the estimated annual cancer incidence was adjusted to reflect the unit risk factor in Table 2-6. In general, there was little net effect on an individual study's overall estimate of cancer cases as a result of this modification (see Table 2-9). The one exception to this was the South Coast study. The decrease in estimated annual cancer cases for the South Coast study was due to large differences between the California Department of Health Services (DOHS) unit risk factors used for several pollutants in that study and EPA's unit risk factors for those pollutants. As seen in Table 2-10, adjusting the South Coast study's estimates of cancer cases by using the unit risk factors in Table 2-6

TABLE 2-8

POLLUTANTS WITH UNIT RISK FACTORS DIFFERENT FROM THOSE USED IN THIS REPORT

	STUDY	POLLUTANTS WITH DIFFERENT UNIT RISK FACTORS[a]
1.	Ambient Air Quality	None
2.	NESHAP/ATERIS	(see footnote b)
3.	Asbestos	None
4.	Coal and Oil Combustion	Beryllium, Formaldehyde
5.	Drinking Water Aerators	EDC, Perchloroethylene, TCE, Vinyl chloride
6.	Gasoline Marketing	None
7.	Hazardous Waste Combustors	BaP, Methylene chloride, Perchloroethylene, TCE, Vinyl chloride
8.	Mobile Sources	Asbestos, Benzene, gasoline vapors, EDB, BaP
9.	Municipal Waste Combustors	None
10.	POTWs	Methylene Chloride, TCE
11.	Radionuclides	-
12.	Sewage Sludge Incinerators	BaP, Cadmium, PCBs, TCE
13.	TSDFs	None
14.	Waste Oil Combustors	TCE, PCBs
15.	35-County	Benzene, BaP, Methylene chloride, TCE
16.	5-City	Benzene, Methylene chloride, TCE
17.	IEMP-Baltimore	Benzene, Perchloroethylene, TCE
18.	IEMP-Kanawha Valley	Benzene, Perchloroethylene, Vinyl chloride, Methylene chloride, TCE, BaP, Allyl chloride
19.	IEMP-Philadelphia	EDC, TCE, Perchloroethylene
20.	IEMP-Santa Clara	Benzene, Gasoline vapors, Methylene chloride, Perchloroethylene, TCE, BaP
21.	Southeast Chicago	Acrylamide, 1,3-butadiene, PCB's, Propylene oxide
22.	South Coast	Benzene, BaP, Chromium, EDB, Methylene chloride, nickel, TCE

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[a] EDC = ethylene dichloride
PCBs = polychlorinated biphenyls
BaP = benzo(a)pyrene
TCE = trichloroethylene

[b] Except for some methylene chloride source categories in the ATERIS data base, all of the unit risk factors in the ATERIS data base are the same as those in Table 2-6. For the NESHAP study as reported in the 1985 Six-Month Study, 21 unit risk factors have changed. The more important one in terms of either annual cancer cases or percent change are: acrylamide, 1,3-butadiene, ethylene dibromide, nickel subsulfide, trichloroethylene, and vinyl chloride. For a complete listing, see Table 3-27.

TABLE 2-9

EFFECT OF CHANGES IN UNIT RISK FACTORS USED IN THIS REPORT ON ORIGINAL ESTIMATES OF ANNUAL CANCER CASES

STUDY	ESTIMATED ANNUAL CANCER CASES	
	Using Risk Factors As Reported in Study	Using Table 2-6 Unit Risk Factors
1. Ambient Air Quality	2,022	2,022
2. NESHAP/ATERIS	504[a]	496[b]
3. Asbestos	82	82
4. Coal and Oil Combustion	11.1	12.1
5. Drinking Water Aerators	0.021	0.021[c]
6. Gasoline Marketing	24-75	24-75
7. Hazardous Waste Combustors	0.3-9	0.3-9[c]
8. Mobile Sources	628-1,874	601-1,852
9. Municipal Waste Combustors	1.7-2.3	1.7-2.3
10. POTWs	1.5	1.3
11. Radionuclides	16	16
12. Sewage Sludge Incinerators	13	13
13. TSDFs	140	140
14. Waste Oil Combustors	0.10-0.56	0.10-0.56
15. 35-County	469-553	463-546
16. 5-City	92.6	90.4
17. IEMP-Baltimore	2.8-7.0	2.95-7.15
18. IEMP-Kanawha Valley	1.8	1.77
19. IEMP-Philadelphia	0.37	0.42
20. IEMP-Santa Clara	2.2	1.85
21. Southeast Chicago	1.21	1.26
22. South Coast	162-221	19-33

NOTE: The reports on Municipal Solid Waste landfills, Superfund sites, and Woodstoves did not include estimates of annual cancer cases.

[a] Based on original NESHAP study as reported in the Six-Month Study.

[b] Incorporates revised NESHAP study estimates and ATERIS data base risk estimates.

[c] The net effect of adjusting unit risk factors cannot be determined as cancer risk attributable to individual organic compounds was not available. The effect is expected to be small.

TABLE 2-10

EFFECT OF UNIT RISK FACTORS ON ESTIMATED ANNUAL CANCER CASES: THE SOUTH COAST STUDY

POLLUTANT	ESTIMATES OF ANNUAL CANCER CASES USING...			
	South Coast Study Unit Risk Factors[a]		EPA Unit Risk Factors[b]	
	Ambient Measured[c]	Model Predicted[c]	Ambient Measured[d]	Model Predicted[d]
Benzene	99	55	16	8.6
Carbon tetrachloride	1.4	0.001	1.4	0.001
Chloroform	1.3	0	1.3	0
Ethylene dibromide	0.37	0.007	1.1	0.02
Ethylene dichloride	-	0.007	-	0.007
Methylene chloride	8.0	3.4	0.92	0.39
Perchloroethylene	0.59	0.43	0.59	0.43
Trichloroethylene	0.33	-	0.43	-
Arsenic	1.5	0.0001	1.5	0.0001
Beryllium	0.09	0.0003	0.09	0.0003
Cadmium	0.49	0.96	0.49	0.96
Chromium	108	102	8.6	8.2
Nickel	0.37	0.09	0.56	0.14
Total Annual Cancer Cases	221	162	33	19

[a] The unit risk factors used in the South Coast study, which are California Department of Health Services' unit risk factors, are found on page V-10 of the South Coast study.

[b] The EPA unit risk factors used to adjust the estimates of annual cancer cases are found in Table 2-6 of this report.

[c] Based on dividing estimated lifetime (70-year) cancer cases in Table VI-3, p. VI-11, of the South Coast study by 70.

[d] For each pollutant, the annual cancer cases in this column were calculated as follows: the estimate of annual cancer cases using the South Coast study's unit risk factors was multiplied by the ratio of the EPA unit risk factor to the California Department of Health Services unit risk factor for that pollutant.

reduced total estimated annual cancer cases by approximately 85 percent. The issue of which unit risk factor, DOHS or EPA, more likely represents actual risk is beyond the scope of this study. For purposes of this study, all cancer risks are evaluated and reported (unless otherwise noted) on the basis of the unit risk factors presented in Table 2-6 and Table 2-7; the unit risk factors from the South Coast study were not used to estimate nationwide cancer risk.

Emission Factors. A second basic reason for different cancer risk estimates is that different pollutant emission factors have been used. Where emission factors could be compared, the most recent emission factor was selected in the calculation of cancer risk. (This selection assumes that the more recently developed emission factor is a better (more accurate) factor than the previous emission factor.) In these instances, appropriate adjustments were made to the cancer risks based on "older" emission factors, and the "new" set of estimated annual cancer incidences per million population were compared. Unfortunately, except for motor vehicles, pollutant emission factors for most source categories were either not readily available in that they were not included in the final report or were reported in only one of the studies, and a comparison could not be made. Thus, it was generally very difficult to say anything about the effect, if any, pollutant emission factors had on discrepant estimated annual cancer incidences per million population.

In several instances, the studies referred to more recently developed emission factors that were used (i.e., the 5-City study) or not used because it was beyond the scope of the study (i.e., the 35-County study). Such qualitative statements were used to some extent in selecting some cancer risk estimates as better than others. In summary, except for motor vehicles, trying to identify differences in emission factors as a source of discrepancy was not very successful.

Modeled vs. Ambient-Measured Concentrations. Cancer risk estimates can also vary depending on whether they are derived from modeled concentrations or from ambient-measured concentrations. Both methods of obtaining ambient concentrations from which cancer risks can be estimated have their own inherent set of limitations (see Table 2-11). It was beyond the scope of this project to analyze the various limitations of the two techniques for estimating ambient concentrations. For example, this study did not try to determine whether the most appropriate models were used in the studies or to try to "correct" the cancer estimates to a single model. Similarly, it was beyond the scope of this project to try to determine whether the proper sampling technique was used to obtain the ambient samples or whether the sampling point locations were likely to obtain representative samples.

The study did, however, attempt to use several guidelines or "thought processes" in evaluating and comparing cancer risks obtained from modeled concentrations and from ambient-measured concentrations. These were:

- Unless otherwise noted in a study, all models were assumed to be appropriate and their results were given equal weight.
- Where modeled and ambient-measured concentrations were used and risk estimates made, an attempt was made to identify potential causes for discrepancies based upon known emission sources. For modeled estimates, this meant trying to identify emission sources included in the inventory and emission sources that were excluded. For ambient-measured concentrations, this meant trying to determine if the locations from which the data were obtained contained known point sources that might influence or bias the data.

TABLE 2-11

SELECTED LIMITATIONS OF MODELED AND AMBIENT-MEASURED CONCENTRATIONS FOR ESTIMATING CANCER RISK

Modeled Concentration Limitations

1. Many of the dispersion models assume flat terrain and average meteorological conditions. Rough terrain in the area surrounding a source, such as a valley, can result in concentrations that are up to one to two orders of magnitude higher or lower than concentrations predicted in gently rolling terrain.

2. Dispersion modeling often extends to only 20 kilometers from the source. This technique can lead to understating risk if extending dispersion increases significantly the number of people exposed.

3. Dispersion modeling estimates are rarely based on site-specific meteorology. Often, data from hundreds of kilometers away must be used.

4. Dispersion models do not consider increases in concentrations that could result from re-entrainment of toxic particles from streets, rooftops, etc. In addition, models do not account for background concentrations, secondary formation of pollutants, and emissions from other sources not explicitly included in the analyses.

5. Emission estimates are generated from data and assumptions that could be in error. For example, although some of the studies (e.g., the 35-County study) incorporate plant-specific emission estimates whenever possible, the pollutant releases for other sources are frequently estimated by applying speciation factors against the volatile organic compound (VOC) and total suspsended particulate (TSP) data in the National Emission Data System (NEDS). Unfortunately, some of the information in NEDS is of questionable consistency and quality for the purposes of quantitative risk assessment.

Ambient-Measured Concentration Limitations

1. A basic limitation is the extrapolation of measurements from a limited number of sites to a much larger geographic area in order to estimate population exposure. This affects both estimating exposure within a city from a limited number of sites to estimate average exposure within the city and estimating nationwide cancer risk from a limited number of geographic areas.

2. Ambient-measured data collected over long periods of time (e.g., at least one year) are frequently unavailable, which limits the ability to make statements as to long-term exposures upon which cancer risk estimates based.

3. All ambient-measured data are subject to errors in sampling and analytical methods.

4. Ambient data may underestimate "true" maximum individual risk (MIR) concentrations because sampling is limited to a small number of fixed monitoring sites.

- In the absence of evidence to the contrary, cancer incidence estimates based on modeled concentrations and those based on ambient-measured concentrations cannot be summed to obtain a total risk estimate (i.e., they are not mutually exclusive). For only one pollutant, 1,3-butadiene, were these two estimates summed. This was done because the locations for the ambient data were not identified as having known point sources of 1,3-butadiene. Therefore, it was felt that a better estimate would be obtained by assuming the ambient data reflected background and area-type emissions of 1,3-butadiene (which would include motor vehicle emissions) to which the cancer risk from the modeled point sources could be added.

- The South Coast study noted several pollutants for which large discrepancies between modeled and ambient-measured concentrations occurred and offered potential reasons for such. Other studies also noted where they believed one methodology may be underestimating risk. In each case, the studies identified the modeled estimate as possibly underestimating risk. The reason most frequently cited for this underestimation was an incomplete inventory of emission sources. Modeling biases can also lead to the underestimation of risk. These discrepancies and their reasons are noted in the pollutant-by-pollutant analysis section found in Appendix B. These reasons were considered in evaluating which risk estimates were "better" than others.

  For formaldehyde and carbon tetrachloride, the ambient-measured concentrations and derived cancer estimates were selected and evaluated. For formaldehyde, this was done because it is well established that formaldehyde is formed in the atmosphere (secondary formation). Ambient-measured data can account for this atmospheric-formed formaldehyde, whereas models do not. In the case of carbon tetrachloride, it is also well known that carbon tetrachloride remains in the atmosphere long after it has been emitted. Thus again, ambient-measured data can account for this "retention" of carbon tetrachloride more readily than models.

- For comparing between ambient-measured data, the geographic coverage of the study was considered. It was assumed that risk estimates based on ambient-measured data from more geographic locations were better estimates from which to estimate nationwide risk than were estimates from single geographic locations. This led to selecting the Ambient Air Quality study results as the best estimates of nationwide risk from those estimates based on ambient-measured data. In fact, most if not all of the smaller geographic ambient data fell within the range of data used in the Ambient Air Quality study.

Source Category Definition and Coverage. One of the basic steps in reducing the data was to determine the various source categories (e.g., motor vehicles, electroplating, municipal landfills) covered by the studies, and then to assign the risk estimates for each pollutant to that source category. This is necessary to avoid double-counting. In this study, specific source categories were used to aggregate and compare the risk data. For most of the source categories and studies, assigning risk estimates to the appropriate source category was relatively easy, as most of the specific source categories were developed on the basis of the source categories reported in the studies. In certain instances, however, it was difficult to determine whether or not a source category in one study was the same as in another. For example, the source categories "heating," "combustion," "residential heating," "coal and oil combustion," and "oil combustion," all appeared in one or more studies. In this instance, it was very difficult to determine whether or not the same types of emission sources were being covered.

Another aspect to source category definition was whether or not the studies included all of or just some (and which ones) of the types of emission sources in a particular source category. For example, some motor vehicle pollutants are exhaust and evaporative emissions as well as tire wear emissions. Some studies reported only the risk from exhaust and evaporative emissions, while one study included those from tire wear. The ability of determining the specific types of sources covered by each study for each source category met with varying success, because the information needed to ensure an accurate accounting was not always reported in the available material. In certain cases, we were able to obtain information beyond that which was published. Thus, assumptions as to which source categories are mutually exclusive or not and whether the same set of emission sources are covered in a particular source category remain, in certain instances, highly uncertain.

Finally, plant location information from the NESHAP/ATERIS data base and various EPA documents was used to determine whether specific plants were located in counties covered by the 35-County study, in the five cities covered in the 5-City study, in the four IEMP study cities, in the South Coast geographic area, and in the Southeast Chicago study area. The relationship of plant locations to the geographic study area of these other studies was used to assess the potential relationship of the risk data (whether they were mutually exclusive and could be added, or whether they were duplicative). Evidence of a match was assumed to infer a likelihood of double-counting if the two risk estimates were added. If no plant location match was found, it was assumed to infer a likelihood of mutual exclusiveness.

Geographic Scope of the Study. As the primary purpose of this study is to evaluate nationwide risk, modeled risk estimates from studies that already have a nationwide scope were generally preferred as better estimates of nationwide risk than those nationwide risks that could be extrapolated from the studies with smaller geographic scopes. This is a somewhat difficult "preference" assumption to make. The smaller localized studies frequently are based on much more detailed and site-specific data than are the nationwide studies. Thus, those studies may do a somewhat better job at estimating likely levels of risk. At the same time, because they take into account site-specific data, they are likely to be less representative of conditions nationwide and thus can not be simply extrapolated nationwide. As this study is in itself a broad scoping type of study, the broader scoping nature of the nationwide studies are more consistent with the goals of this study. Therefore, based upon these considerations, the results of the nationwide studies received preference in developing point estimates of cancer risk.

As noted earlier in this chapter, risk estimates for a few source categories were available only from the smaller, localized studies. Nationwide risks were extrapolated from these studies in some instances (e.g., petroleum refineries). In other instances, so little was known about the emission source that the cancer risk from only that study was used in estimating the total nationwide risk. Such sources are part of one of the general source categories (e.g., unspecified sources, miscellaneous).

As noted earlier, for ambient-measured risk estimates, those from the Ambient Air Quality study were generally assumed preferable to those extrapolated from the smaller, localized studies because of its broader geographic scope.

Miscellaneous Specific Considerations. As the studies and various risk estimation methodologies were reviewed, several additional factors were considered in evaluating the data.

- The 35-County study noted that the counties studied were selected, in part, because of the presence of known emission sources of the pollutants being considered. Thus, the estimates of annual cancer incidence per million population calculated for the 35-County study may be higher than the nationwide population-weighted average. Applying the 35-County study's rates directly to the total U.S. population could result in an overestimation of cancer risk.

- Several methodologies exist for estimating risk from PIC. Each methodology has its own inherent limitations, and no methodology has been shown to be better than another. The current trend in estimating risk has been toward using individual source category emission factors and developing unit risk factors that are based upon the mixture of components emitted from the source category. For purposes of this study, the modeled estimates of risk from PIC were selected from those estimates using this type of risk estimation methodology.

Limitations and Uncertainties

Limitations

Consistent with the purposes of this study, the analyses in this report consider cancer risk only from air toxics. Noncancer effects of air toxics are not included. As noted in Chapter 1, other studies are being undertaken to examine other health effects, such as subtle biochemical, physiological, or pathological effects to gross effects, including death.

The only pathway considered in this report is inhalation. Potential health risks from ingestion of air pollutants that ultimately reach humans through the diet or that are directly ingested are not examined. Neither are the potential environmental effects of direct deposition and urban runoff of air pollutants to surface water addressed.

Estimates of cancer risk are based on concentrations of air toxics found in the ambient air. It was not the purpose of this study to estimate cancer risk based on exposure to indoor concentrations of air toxics. As noted in Chapter 1, a separate program has been initiated to quantify the risk from indoor exposure to air toxics.

Although quantitative risk estimates are reported in this study, it is important to remember that the reports and studies used do not cover either all known or potential air toxics or all sources of air toxics which contribute to outdoor exposure. As noted earlier in this chapter, the cancer risk estimates in the reports and studies reviewed cover 90 compounds in approximately 65 source categories. These compounds represent only a fraction of the total number of compounds present in the ambient air. Based on a review of studies directed at identifying compounds in the ambient air, more than 2,800 compounds have been identified as existing in the atmosphere,[9] some of which may be toxic at ambient levels. One major factor preventing analysis of more pollutants is the lack of measurement techniques to obtain ambient measurements for a number of pollutants. A second major factor is the lack of data on cancer risk associated with ambient concentrations of other pollutants. Only about 10 percent (approximately 300) of the 2,800 plus atmospheric pollutants have been tested for mutagenicity or carcinogenicity. Of these, 97 have tested positive in whole animal bioassays. The mutagenicity or carcinogenicity of the other 2,500 atmospheric compounds is unknown. The impact on cancer incidence from these other atmospheric compounds is currently impossible to estimate.

Despite the fact that more than 2,800 chemicals have been identified in ambient air, a large number of unknown compounds are still likely to exist. Indeed, atmospheric chemists studying the reactions of most common urban pollutants are often able to account for only about one-half of the carbon in their studies. The impact of the unidentified organic products on cancer incidence is unknown. However, the compounds for which risk information is available were selected based on evidence that led to their being suspected carcinogens. Thus, it is possible that the cancer risk associated with the 90 or so compounds for which cancer risk data have been obtained represents a much larger proportion of the total risk than might be suggested by a simple comparison of the 90 compounds to the total number of atmospheric compounds.

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[9] Graedel, T.E., D.T. Hawkins, L.D. Claxton. Handbook of Atmospheric Compounds: Sources, Occurrence, and Bioassay. HERL-051a. (1985: Academic Press, New York).

The quantitative risks reported in this study are based, in part, on unit risk factors that are either upper-bound estimates or maximum likelihood estimates of the carcinogenicity potential of the air toxic pollutants. In either case, the quantitative estimates based on these unit risk factors are conservative in that actual cancer cases for these pollutants may be higher, but are more likely to be lower than the estimates presented in this study. Thus, the aggregate cancer risk, which is based on the summation of individual pollutant's cancer risks, represents a likely overestimate for those pollutants considered.

The amount and quality of information concerning pollutants and their risk from specific source categories vary considerably. For example, information on the types of pollutants emitted from motor vehicles is fairly well established. In addition, emission factors for most motor vehicle pollutants have been estimated much more closely than for other source categories because, in part, of the relative ease with which motor vehicles can be tested. On the other hand, the types of pollutants from source categories, such as TSDFs and Superfund sites, are much more likely to vary because the materials that give rise to the pollutants vary from one site to another. Also, the emission levels of pollutants from such source categories are much more difficult to establish because the test methodologies are not as easy to apply as those for motor vehicles. It should be noted that there is considerable uncertainty associated with the estimates of risk attributed to individual pollutants emitted from TSDFs. It is possible that dioxin, the TSDF pollutant for which the largest risk is estimated, may be emitted in much smaller quantities, if at all, from TSDFs. Finally, estimates of risk for some source categories may suffer simply from a lack of a complete accounting of pollutants.

Reliable quantitative estimates remain unavailable for many potentially important source categories (e.g., Superfund sites) and, in some instances, sources may have been missed in the source assessment. In addition, quantitative estimates of risk from pollutants formed or transformed in the atmosphere (secondary formation) remain unquantified for almost all pollutants. The most important secondary pollutant for which cancer risks have been quantified to date is formaldehyde. Formaldehyde is both emitted directly into the atmosphere and formed in the atmosphere, and atmospheric formation of formaldehyde has the potential to produce many times the amount directly emitted from most sources. The gas-phase transformation products of a variety of common urban pollutants and air toxics have been shown to be potentially hazardous. The normal atmospheric reactions of these pollutants produce a variety of oxygenated and nitrogenated products, such as glyoxal and peroxyacetylnitrate (PAN), and a variety of unidentified species, which have been shown to be mutagenic. The total mutagenicity of the transformation products is often many times greater than the mutagenicity of the original pollutants. The fact that a gas-phase product is mutagenic in a bacterial test system suggests, but does not establish, that a human health risk may arise from exposure to such products. It is not currently possible to quantify the risk from exposure to the unidentified, potent gas-phase mutagens produced in these photochemical reactions. Nevertheless, the evidence to date clearly suggests that the transformation of ubiquitous, often innocuous, urban pollutants may add a significant additional risk component to any assessment of urban exposure and risk.

Uncertainties

There are many uncertainties associated with the methodologies that are available for making the risk estimate used in the reports and studies that formed the data base for this report. The following uncertainties are among the more important ones to keep in mind. The list is not all inclusive, and additional uncertainties are identified throughout the report.

- Cancer incidences presented in this report are based on the assumption that emission levels and ambient levels for each pollutant either "averages out" over a 70-year period to equal the concentrations used in the calculations of annual incidence or remain constant for that period of time. In reality, emissions and air quality will vary from year to year. Because the amount and direction of variation is unknown, it is unclear how much this assumption affects the results.

- All of the analyses assume exposure to air toxics occurs where people reside. This assumption does not consider the possibility that people may move throughout the urban area and change their homes several times during their lives. In addition, few plants may operate or be expected to emit at the same level for 70 years, though the areas in which they are located may remain industrial. Thus, future exposures may be either worse or better than the old environment. Because exposures are simulated over a 70-year period, it is unclear how much this assumption affects the results.

- All of the risks assume continuous outdoor exposure. This assumption ignores the fact that people spend the majority of their time indoors, and thus are exposed to indoor atmospheres, which can be significantly different from the outdoor atmosphere. Indoor concentrations of certain pollutants (e.g., radon, tobacco smoke, formaldehyde, and other VOCs) are commonly several times higher than outdoor concentrations. Estimated cancer risk to such indoor pollutant concentrations suggest that cancer risks based solely on outdoor exposure may be understated for such pollutants. On the other hand, the extent to which certain pollutants (e.g., trace metals) penetrate indoors is large unknown. If emissions of a pollutant do not penetrate completely indoors and if there are no indoor sources of that pollutant, then cancer risks based solely on outdoor exposure will have been overstated.

- All risks are assumed to be additive. This assumption can lead to substantial errors in risk estimates if synergistic or antagonistic interactions occur. Although dose additivity has been shown to predict the acute toxicities of many mixtures of similar and dissimilar compounds, some marked exceptions have been noted. Consequently, additivity assumptions may substantially overestimate risk in some cases and underestimate it in others. The available data on mixtures are insufficient for estimating the magnitude of these errors. Based on current information, additivity assumptions are plausible for component compounds that induce similar types of effects at the same sites of action.

- Unit risk factors used in this study have been generated, in most instances, using EPA approaches or models. Most of the resulting unit risk factors are generally regarded either as plausible, upper-bound estimates or as maximum likelihood estimates. The linearized multistage procedure used to derive these factors leads to a plausible upper limit to the risk that is consistent with some proposed mechanisms of carcinogenesis. Such estimates, however, do not necessarily give a realistic prediction of the risk. The true value of the risk is unknown, and may be as low as zero.

- Cancer unit risk values are subject to much uncertainty and in many cases are preliminary estimates. The risk estimates in the reports are based on layers of assumptions concerning the health effects of chemicals, the degree of human exposure, and the way these substances interact inside the human body. For example, the weight of evidence of carcinogenicity for the compounds identified in this report varies greatly, from very limited to very substantial. Further, the extent of evaluation and health review performed varies considerably among compounds. As additional scientific information is acquired, these values could change significantly, as they have in the past, and thus the magnitudes and relative importance of particular pollutants can change.

- In developing its unit risk factors, EPA uses a nonthreshold, multistage model, which is linear at low doses, to extrapolate from high-dose response data to the low doses typically caused by exposure to ambient air. In other words, carcinogenic substances are assumed to cause some risk at any exposure level. If the true dose-response relationship at low doses is less than linear, then the unit risk estimates err on the high side.

- Many of the individual pollutants have specific uncertainties that affect their potential contribution to cancer risk. Chapter 3 and Appendix B identify these uncertainties. Chromium, formaldehyde, and PIC are three of the major contributors, based on this study, to cancer risk. Each have specific uncertainties that may significantly affect the estimate of cancer risk attributed to them. These uncertainties are highlighted below.

  In the case of chromium, only the hexavalent form has been proven to be carcinogenic. The percentage of total chromium that is hexavalent is known to vary considerably depending on the source. For example, hexavalent chromium is less than 1 percent of total chromium emissions from coal and oil burning combustion, while it is nearly 100 percent of total chromium emissions from cooling towers and electroplating. Nevertheless, considerable uncertainty remains as to the exposure to hexavalent chromium versus total chromium emissions.

In the case of formaldehyde, a number of unresolved issues concerning its carcinogenesis have been the subject of a considerable amount of scientific debate. At the center of this debate are questions concerning such issues as the mechanism of action of formaldehyde at the molecular level, the shape of the dose-response curve, the importance of irritation and the role of the mucus blanket, and the significance of endogenous formaldehyde. The EPA has determined that the 95-percent upper confidence limit on risk for formaldehyde, based on data from a 24-month animal study conducted by the Chemical Industry Institute of Toxicology (CIIT), is the appropriate statistical estimate to use in assessing human risk. This is consistent with the EPA Guidelines for Carcinogen Risk Assessment, which state that in the absence of compelling biological information on the mechanism of action, the linearized multistage procedure should be used to derive an upper bound estimate of risk. The EPA does not recommend the use of maximum likelihood estimates of cancer risk based on animal data; such estimates are highly unstable (i.e., small changes in the data may cause orders-of-magnitude fluctuations in the risk estimates). The EPA is currently evaluating new scientific data on formaldehyde and will publish an update to the 1987 assessment at some time in the future.

There has also been disagreement over whether to consider the incidence of both malignant and benign tumors in rats or whether only the malignant tumors are significant. The unit risk factor based on total tumors is approximately 14 times higher than the unit risk factor based on malignant tumors only. The current consensus is that only the malignant tumors should be used to assess the human cancer risk from formaldehyde. There appears to be little evidence that benign tumors progress to any of the malignant tumors seen in the CIIT study. The unit risk factor based on malignant tumors only is used in this report to estimate cancer incidence from exposure to formaldehyde.

In the case of PIC, there are several sources of uncertainty. There are a number of methodologies available to estimate risk from PIC. Some of these methodologies use BaP as a surrogate for both PIC emissions and unit risk value. Others use PIC-specific emission factors and unit risk factors or comparative potency factors. The estimate of cancer incidence is seen to vary by a factor of 200 depending on which methodology is used. While no one methodology has been shown to be a better methodology for estimating risk from PIC, this study uses the methodology that relies on PIC-specific emission factors and unit risk factors or comparative potency factors.

The unit risk factors for estimating risk from PIC are of particular concern. As noted previously in this chapter, many of the unit risk factors used in this study have been approved by EPA. The most important exception to the use of EPA approved unit risk factors are the group of compounds known as PIC. There is no current EPA unit risk factor for this group, although unit risk numbers are available for some of the compounds (e.g., BaP) that compose PIC. The 1985 Six-Month Study used a unit risk factor of 4.2×10^{-1} $(\mu g/m^3)^{-1}$ for PIC. This unit risk factor was derived in a highly unusual manner, and represents an initial attempt at quantifying the potential risk from PIC. Any estimate based upon this unit risk factor is highly tentative.

Other unit risk factors for estimating the cancer risk from PIC have become available more recently. These unit risk factors represent estimates of risk from PIC mixtures emitted from specific source categories (e.g., motor vehicles, hazardous waste incinerators). Some of the more recent unit risk factors were estimated using what is known as the comparative potency approach.[10] Even though the more recent factors are also uncertain and have not received the same level of scrutiny by EPA as for other unit risk factors, it was felt that they were an improvement over the PIC unit risk factor used in the 1985 Six-Month Study. Thus, the risk estimates from PIC presented in this report reflect the use, where possible, of the more recently developed unit risk factors for specific PIC mixtures.

Another source of uncertainty associated with PIC is the selection of the appropriate unit risk factor for diesel particulates, which are included with this group of compounds. Unit risk factors identified for diesel particulates range from 2×10^{-5} to 1×10^{-4} $(\mu g/m^3)^{-1}$. The EPA has not yet determined a single best estimate of the unit risk factor for these particulates. Thus, the estimate of risk from all sources of PIC includes the range of risk created by the range in the unit risk factor for diesel particulates.

Major uncertainties exist for many other chemicals addressed in this report. For example, there is considerable debate in the scientific community concerning the mechanism of carcinogenic action and the estimation of cancer potency for dioxin. Another unresolved issue concerns the relevance to man of the kidney pathology observed in rats following exposure to gasoline vapors. A detailed discussion of the uncertainties associated with risk estimates for these and other chemicals is outside of the scope of this report.

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[10] For a brief discussion on the comparative potency approach, see page B-110 of Appendix B.

In summary, the portion of the entire cancer risk represented by pollutants and source categories not covered in this study is unknown. It is expected that the pollutants and source categories covered are among the most likely major contributors to cancer risk based on our current state of knowledge regarding carcinogenicity of pollutants and sources that emit those pollutants. As new information is obtained, other pollutants and sources may be found to be as important, or even more important, contributors to cancer risk.

As a result of the limitations and uncertainties identified above, the numerical estimates presented in this report should be viewed only as a rough indication of the potential for cancer risk caused by a limited group of pollutants found in the ambient air. Many of the risks cited in this report are almost certainly inaccurate in an absolute sense. The best use of the risk estimates is in describing the broad nature of cancer risk posed by these air toxics and by making relative comparisons of risks across pollutants and sources.

CHAPTER 3.0

THE MAGNITUDE AND NATURE OF THE CANCER RISK

In this study, the magnitude and nature of the air toxics problem were evaluated based upon the results of a diverse collection of reports and studies. These reports and studies cover many pollutants and source categories. They also cover varying geographic areas, ranging from city-specific studies to nationwide studies. The methodology used to estimate the magnitude and nature of the cancer risk nationwide from this diverse collection of reports and studies was described in the previous chapter.

In this chapter, the overall magnitude of the cancer risk is presented first. The magnitude of the cancer risk is presented in terms of annual cancer cases and lifetime individual risk. The nature of the cancer risk problem is then described in terms of individual pollutants, source categories, and geographic variability. Finally, the results of this study are compared with those of the 1985 Six-Month Study.

It is important to understand that these estimates reflect the use of either an upper bound or a maximum likelihood estimate of unit risk; that is, for the pollutants examined, the actual cancer risk may be higher but is more likely to be lower. As discussed in Chapter 2, this occurs because of the manner in which EPA calculates the unit risk factors for toxic pollutants.

Magnitude of the Cancer Risk Problem

The magnitude of the cancer risk is presented first in terms of total nationwide cancer cases per year and then in terms of lifetime individual risk. Both measures of the magnitude of cancer risk play an important role in the understanding of the problem and in the development of air toxic control strategies and regulations. Detailed analyses for those pollutants that were initially identified as potentially resulting in ten or more cancer cases per year nationwide are found in Appendix B.

Annual Cancer Cases

The estimates of nationwide annual cancer cases for 26 specific pollutants are presented in Table 3-1. The remaining pollutants are grouped together under "Miscellaneous." Annual cancer incidence was calculated by dividing the estimated lifetime incidence levels by 70 years.[1]

Both range and point estimates of nationwide annual cancer cases are presented in Table 3-1. These estimates were derived, in most instances, from annual cancer incidence estimates based on both modeled and ambient-measured concentrations. The estimates under the column "Range" reflect a narrowing of the total range of nationwide annual cancer incidence that can be calculated from the various studies. As seen in Table 3-1, the range of estimates is about two-fold in size, being approximately 1,400 to 2,900 cancer cases per year.

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[1] The unit risk factors used in this study represent the chance of contracting cancer from a lifetime (70 years) exposure to a given concentration of that pollutant. It was assumed that the resulting lifetime incidence levels could be divided by 70 to represent annual incidence levels.

TABLE 3-1

SUMMARY OF ESTIMATED NATIONWIDE ANNUAL CANCER CASES BY POLLUTANT

		ESTIMATED ANNUAL CANCER CASES[a]	
POLLUTANT	EPA CLASSIFICATION[b]	RANGE	POINT[c]
1. Acrylonitrile	B1	13-14	13
2. Arsenic	A	8-68	68
3. Asbestos	A	82-126	88
4. Benzene	A	143-181	181
5. 1,3-Butadiene	B2	244-266	266
6. Cadmium	B1	6-16	10
7. Carbon tetrachloride	B2	31-47	41
8. Chloroform	B2	29-115	115
9. Chromium (hexavalent)	A	113-283	147-265[d]
10. Coke Oven Emissions	A	7-11	7
11. Dioxin	B2	2-125	2-125[e]
12. Ethylene dibromide	B2	25-68	68
13. Ethylene dichloride	B2	16-45	45
14. Ethylene oxide	B1-B2	5-6	6
15. Formaldehyde	B1	124-240	124
16. Gasoline vapors	B2	19-76	19-76[f]
17. Hexachlorobutadiene	C	9	9
18. Hydrazine	B2	6	6
19. Methylene chloride	B2	3-6	5
20. Perchloroethylene	B2	6-13	6
21. PIC[g]	-	438-1120	438-1120[h]
22. Radionuclides[i]	A	1-3	3
23. Radon[i]	A	2	2
24. Trichloroethylene	B2	5-13	7
25. Vinyl chloride	A	13-25	25
26. Vinylidene chloride	C	0.5-10	10
27. Miscellaneous[j]	-	15	15
Totals		1,366-2,909	1,726-2,706

NOTE: Values in this figure are not absolute predictions of cancer occurrence and are intended to be used in a relative sense only. The dose-response relationships and exposure assumptions have a conservative bias, but omissions due to uncharacterized pollutants (either directly emitted or secondarily formed) and emission sources, the long-range transport of pollutants, and the lack of knowledge of total risk from multi-pollutant exposures will offset this bias to an unknown extent.

[a] These estimates are based on unit risk factors that may overstate the actual risk. The unit risk factors for arsenic, benzene, cadmium, and hexavalent chromium are maximum likelihood estimates. The unit risk factor for

FOOTNOTES TO TABLE 3-1 (continued)

asbestos is a "best" estimate, very similar to the value that would be obtained if the maximum likelihood estimate was calculated. Unit risk factors for PIC have no classification. All other unit risk factors are upper-bound estimates.

[b] For a discussion of how EPA evaluates suspect carcinogens and more information on these classifications, refer to "Guidelines for Carcinogen Risk Assessment" (51 Federal Register 33992). The EPA classifications used in this report are:

A = proven human carcinogen

B = probable human carcinogen (B1 indicates limited evidence from human studies and sufficient evidence from animal studies; B2 indicates sufficient evidence from animal studies but inadequate evidence from human studies)

C = possible human carcinogen

[c] If a range is shown, it was considered unreasonable to select a point estimate.

[d] Range primarily reflects uncertainty with the exposure to hexavalent chromium from cooling towers. Some uncertainty to actual exposure to hexavalent chromium from all sources exists because the percent of total chromium that is hexavalent is still being evaluated for most sources.

[e] Range reflects great uncertainty associated with exposure to dioxin from treatment, storage, and disposal facilities, and from municipal waste combustors. Other uncertainties associated with dioxin estimates include sampling method and extrapolation from tetrachlorinated dibenzodioxin (TCDD) to the other dioxin subspecies.

[f] Range reflects different assumptions as to which portion of gasoline vapors is carcinogenic. The upper end of the range assumes all gasoline vapor is carcinogenic; the lower end assumes only the C6 and higher fraction of the gasoline vapor is carcinogenic.

[g] PIC (products of incomplete combustion) is a group of pollutants that have not been very well defined and for which EPA has not developed a classification. It is composed of some pollutants, such as BaP, for which EPA has developed a classification. BaP is a B2 pollutant (probable human carcinogen).

[h] Range reflects the use of two unit risk factors for diesel particulates.

[i] From sources emitting significant amounts of radionuclides (and radon) to outdoor air. Does not include exposure to indoor concentrations of radon due to radon in soil gases entering homes through foundations and cellars.

[j] Includes individual pollutants primarily from the TSDF study and the Sewage Sludge Incinerator study.

The estimates under the column entitled "Point" reflect an attempt to derive a single estimate of nationwide annual cancer incidence. For most pollutants, a reasonable point estimate could be selected. Point estimates are reported in this column when either only one study reported that pollutant (and as a single point estimate) or the analysis for that pollutant (see Appendix B for discussion) suggested that a single point estimate was a better indicator of risk than a range. For four pollutants, as discussed below, this could not be done. For these four pollutants, a narrower range was estimated. As seen in Table 3-1, the "point" estimates narrow the overall range slightly, to between approximately 1,700 and 2,700 cancer cases per year nationwide. Between 25 and 40 percent of this range is attributable to the cancer risk estimated for products of incomplete combustion (PIC). As noted in Chapter 2, there are many uncertainties associated with estimates of cancer risk from PIC.

As noted above, a point estimate was not reasonable for four pollutants. These four pollutants are PIC, dioxin, gasoline vapors, and hexavalent chromium. In the case of PIC, the large range is created primarily by the uncertainty of the unit risk factor associated with diesel particulates, which are included in the estimates of risk for PIC. A single unit risk factor has not been identified by EPA's Office of Research and Development for diesel particulates. Instead, a range of unit risk factors, from 1.0×10^{-9} to 2.0×10^{-5} $(\mu g/m^3)^{-1}$, has been identified by EPA's Office of Mobile Sources. This range was used to estimate the cancer risk from diesel particulates and is reflected in the total estimated cancer risk from PIC.

In addition, uncertainty lies in the methodologies available for estimating risk (as discussed in Chapter 2 and Appendix B) for PIC. One methodology uses benzo(a)pyrene (BaP) concentrations and BaP unit risk factors to estimate risk from PIC. This methodology assumes BaP is the only carcinogenic component of PIC. More recently developed methodologies use source-specific PIC emission factors and unit risk values to estimate risk. None of the methodologies have undergone a high degree of scrutiny at this time. Thus, although this report uses the more recently developed methodologies to estimate cancer risk from PIC, sufficient uncertainty remains concerning all methodologies associated with PIC risk estimation such that a range might still have been selected as the reasonable best estimate for PIC, even if a single unit risk factor could be identified for diesel particulates.

The range for dioxin is the result of difficulties with the sampling methodologies used to estimate emissions of dioxin and with the methodology used to extrapolate risk from tetrachlorinated dibenzodioxin (TCDD) to the other dioxin subspecies. In addition, much of the range is the result of the uncertainty associated with the risk of dioxin from treatment, storage, and disposal facilities (TSDFs) for hazardous waste. Although the TSDF study allows the calculation of a single point estimate (of 91 cancer cases per year), the underlying emissions data are very uncertain. Actual cancer cases attributable to dioxin emissions from TSDFs could be considerably less. Finally, early data on municipal waste combustors (MWCs) showed a wide range of estimated annual cancer cases (approximately 2 to 20). Recent revisions to the MWC study suggest that the estimated risk attributable to dioxin may be one-half this estimate. For these reasons, no attempt was made to develop a point estimate for dioxin.

For gasoline vapors, the range in estimated risk reflects the uncertainty over quantifying the emissions that are associated with the cancer-causing portion of gasoline vapors. It has been suggested that only a portion (i.e., only those C_6 and higher components), rather than all, of total gas vapors are carcinogenic. At this time, it is uncertain as to which provides a better estimate of the emissions of concern.

For chromium, the range reflects uncertainty over the ratio of hexavalent chromium to total chromium emissions for various chromium emission sources. Several studies (e.g., the 5-City study) attempt to consider available information on the estimated ratios of hexavalent chromium to total chromium for specific sources. For cancer risk estimates based on ambient-measured concentrations of chromium, estimating cancer risk is complicated by the fact that the sources that contribute to the ambient measured chromium concentrations are not identified. Thus, estimating what fraction of total measured chromium may be hexavalent is even more difficult and uncertain. This degree of uncertainty makes any single estimate untenable, and therefore a range has been retained at this time.

Lifetime Individual Risk

In addition to annual incidence, the magnitude of cancer risk from air toxics can be described in terms of an individual's lifetime risk. The lifetime individual risk is a measure of the probability that an individual will develop cancer as a result of exposure to the ambient concentration of an air pollutant over a lifetime (i.e., a 70-year period).[2] The ambient concentration used to calculate lifetime individual risk may be measured or model-predicted. Lifetime individual risk at a particular location is calculated by multiplying the unit risk factor by the estimated long-term average exposure at that location. Where the average ambient concentration is representative of an entire geographic locale (e.g., a city), the term "areawide" or "urban-wide" lifetime individual risk can be used.

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[2] Lifetime individual risk is calculated as follows:

Lifetime individual risk = (exposure concentration) x (unit risk factor)

Frequently, the lifetime individual risk is reported as "maximum individual risk" (MIR). Maximum individual risk refers to an estimate of the maximum level of lifetime individual risk to which a person could be exposed. The MIR is calculated at the specific location near an emission source where the highest long-term average concentration is predicted. It is best characterized, especially when developed as part of preliminary risk assessments, as a rough measure of the potential maximum individual lifetime cancer risk associated with exposure to the maximum modeled long-term concentration. The MIR is not an appropriate measure of the risk level affecting the entire population residing near a particular facility, but rather only to the individuals residing at the specific point of estimated maximum exposure.

The highest predicted modeled concentration may or may not always occur at a point where an individual actually lives. When the highest predicted modeled concentration is found to occur in an inhabited area, the term "maximum exposed individual" (MEI) may be used to refer to the maximum individual risk to which an individual is exposed.

Highly spatially-resolved models are recommended for calculating MIRs. These models, such as EPA's HEM-SHED, calculate individual risks close in (<1 kilometer) for all potential receptor locations around specific point sources. Some model-based studies, however, use a larger spatial setting of grid cells, and thus may not identify the highest modeled concentrations. Similarly, measured concentrations are unlikely to identify the highest concentrations because of the too few monitoring sites generally used in most studies. Thus, in monitoring-based studies and model-based studies, it is often more appropriate to refer to the "maximum individual risks" reported as either "highest observed individual risks" or "highest grid-cell individual risks," respectively.

For purposes of this study, the term "maximum individual risk" (MIR) is used to refer to the highest lifetime individual risk reported in the various studies and reports. MIRs were estimated for individual sources (e.g., waste oil combustors, POTWs), individual pollutants (e.g., arsenic, benzene), and locations (e.g., traffic intersection, geographic locale). MIRs for individual sources reflect the aggregate risk associated with multiple pollutants emitted from that source. MIRs for individual pollutants reflect the risk for that pollutant either from an individual plant within a particular source category (e.g., waste oil combustors) or from sources across multiple source categories (e.g., the Ambient Air Quality study). MIRs for locations reflect the aggregate risk associated with multiple pollutants and sources.

Table 3-2 summarizes the maximum individual risks reported in the various studies for individual pollutants and facilities. Almost all of the studies reported maximum individual risks of at least 1.0×10^{-4}. Many studies showed maximum individual risks of 1.0×10^{-3} or higher. Where appropriate and where possible, footnotes are used to further clarify the types of lifetime individual risks and the procedures used to calculate them.

TABLE 3-2

SUMMARY OF MAXIMUM INDIVIDUAL RISKS OF CANCER AS REPORTED IN THE VARIOUS STUDIES

STUDY	INDIVIDUAL SOURCE/ POLLUTANT/LOCATION	MAXIMUM LIFETIME INDIVIDUAL RISK	REFERENCES
Waste Oil Combustion	Individual source[a]	1.8×10^{-4}	1
	Arsenic	1.6×10^{-4}	
	Cadmium	2.1×10^{-5}	
Hazardous Waste Combustors	Individual source[b]	$<1 \times 10^{-7}$ to 1×10^{-4}	2
TSDF	Individual source[c]	2×10^{-2}	3
Sewage Sludge Incinerators	Individual source[d]	5×10^{-2}	4
Municipal Waste Combustors	Individual source[e]	10^{-5} to 10^{-3}	5
POTW	Individual source[f]	4.5×10^{-2}	6
Coal and Oil Combustion	Individual source[g]	7×10^{-5}	7
	Arsenic	4×10^{-5}	
	Beryllium	2×10^{-5}	
	Cadmium	1×10^{-5}	
	Hexavalent chromium	8×10^{-7}	
	POM	5×10^{-6}	
	Formaldehyde	1×10^{-7}	
Drinking Water Aerators	Individual source[b]	2×10^{-8} to 2×10^{-5}	8
Asbestos[h]	Fabricating	2×10^{-3}	9
	Milling	3×10^{-5}	
	Renovation		
	removal	6×10^{-7}	
	dispose	3×10^{-5}	
	Demolition		
	removal	4×10^{-5}	
	disposal	7×10^{-3}	
South Coast	Benzene	10^{-4} to 10^{-3}	10
	Hexavalent chromium	10^{-4} to 10^{-3}	
Southeast Chicago	Grid cell (populated)[i]	9×10^{-4}	11
IEMP-Baltimore[j]	Benzene	1.0×10^{-4}	12
	Chloroform	1.1×10^{-4}	
	Hexavalent chromium	$<3.6 \times 10^{-4}$	
	Five others	$<6.8 \times 10^{-5}$	
IEMP-Santa Clara[k]	Traffic intersection	3×10^{-4}	13
	Benzene	2×10^{-4}	
	Ethylene oxide	2×10^{-4}	

TABLE 3-2 (concluded)

SUMMARY OF MAXIMUM INDIVIDUAL RISKS OF CANCER AS REPORTED IN THE VARIOUS STUDIES

STUDY	POLLUTANT/SOURCE CATEGORY	MAXIMUM LIFETIME INDIVIDUAL RISK	REFERENCES
IEMP-Kanawha Valley[l]	Institute	8×10^{-3}	14
IEMP-Philadelphia	Chemical mfg.	2.2×10^{-4}	15
Ambient Air Quality[m]	PIC	8.4×10^{-3}	16
	Arsenic	3.9×10^{-4}	
	Cadmium	3.3×10^{-4}	
	Hexavalent chromium	3.7×10^{-4}	
	Chloroform	6.4×10^{-4}	
	Benzene	1.7×10^{-4}	
	1,3 butadiene	1.3×10^{-4}	
	Carbon tetrachloride	5.2×10^{-5}	
	Ethylene dibromide	7.9×10^{-5}	
	Ethylene dichloride	1.1×10^{-4}	
	Formaldehyde	3.0×10^{-4}	
	Methylene chloride	9.6×10^{-6}	
	Styrene	1.8×10^{-6}	
	Perchloroethylene	1.2×10^{-5}	
	Trichloroethylene	1.2×10^{-5}	
	Vinyl chloride	1.0×10^{-5}	
	Vinylidene chloride	1.7×10^{-5}	
NESHAP/ATERIS Data Base[n]	Acetaldehyde	5.0×10^{-4}	17
	Acrylonitrile	3.8×10^{-3}	18
	Arsenic	1.2×10^{-2}	17
	Benzene	6.0×10^{-3}	19
	Beryllium	1.9×10^{-5}	17
	Butadiene	3.2×10^{-1}	17
	Cadmium	1.2×10^{-5}	17
	Carbon tetrachloride	5.7×10^{-3}	17
	Chloroform	2.0×10^{-2}	17
	Hexavalent chromium	1.8×10^{-3}	17
	Coke oven emissions	3.4×10^{-2}	20
	Ethylene dichloride	1.1×10^{-2}	17
	Epichlorohydrin	1.6×10^{-4}	17
	Ethylene oxide	2.6×10^{-2}	17
	Hexachlorobenzene	1.4×10^{-5}	21
	Formaldehyde	9.8×10^{-5}	17
	Methylene chloride	4.0×10^{-3}	17
	Perchloroethylene	5.4×10^{-5}	17
	p-dichlorobenzene	2.8×10^{-4}	17
	Styrene	5.2×10^{-4}	17
	Trichloroethylene	8.1×10^{-5}	17
	Vinyl chloride	6.5×10^{-5}	17
	Vinylidene chloride	1.3×10^{-3}	17

FOOTNOTES TO TABLE 3-2

[a] The MIRs for the individual source and the pollutants are assumed representative of the entire population of waste oil combustors.

[b] Range covers individual MIRs for each modeled facility in the source category.

[c] MEI to "highest annual average ambient concentration around a TSDF."

[d] Based on 10th percentile of all sewage sludge incinerator test data for a non-specified facility.

[e] For existing facilities. Range associated with MIRs at different types of municipal waste combustors.

[f] MIR is associated with one specific POTW. Other POTWs have lower MIRs.

[g] MIRs were calculated for three types of boilers (industrial, commercial, utility) and two types of firing (oil-fired and coal-fired) for each type of boiler. This MIR is associated with an oil-fired, commercial boiler. The range of MIRs estimated was from 2×10^{-6} (oil-fired, utility boiler) to 7×10^{-5} (oil-fired, commercial boiler). The MIRs for the individual pollutants are associated with oil-fired commercial boilers except for POM (coal-fired commercial boiler) and radionuclides (coal-fired industrial and utility boilers). For additional information, see Appendix C, page C-10.

[h] MIR not absolute maximum, but reasonable estimate of highest risk expected.

[i] An MIR of 5×10^{-3} was estimated for a grid cell, but census data indicated that no one was living in that grid cell.

[j] Based on highest average value reported for the pollutant at any of the monitoring sites.

[k] MIR for the traffic intersection is associated with risks from four pollutants. The MIR for benzene is based on maximum concentration at a traffic intersection. The MIR for ethylene oxide is based on maximum concentration at a hospital.

[l] Site of MIR is near a specific facility in Institute, WV, and is based on exposure to six pollutants.

[m] Based on highest arithmetic mean concentration observed.

[n] The lifetime individual risks from the ATERIS database are highly uncertain. The ATERIS contains data from all stages of air toxics analyses, from the very preliminary to the more detailed.

REFERENCES TO TABLE 3-2

1. Peters, W., Duggan, G., and R. Fegley. Waste Oil Combustion Cancer Risk Assessment. Technical Staff Paper. October 1987. page 3.

2. U.S. EPA, Office of Solid Waste. Regulatory Impact Analysis for Hazardous Waste Boilers and Industrial Furnaces. Draft. Exhibits 7-6, 7-9, 7-12, and 7-14.

3. U.S. EPA, OAQPS. Hazardous Waste TSDF - Background Information for Proposed RCRA Air Emission Standards. Volume 1 - Chapters. Preliminary Draft EIS. March 1988. p. 6-10.

4. U.S. Environmental Protection Agency. Standards for the Disposal of Sewage Sludge. Proposed Rule. February 6, 1989. 54 FR 5783.

5. U.S. EPA, Office of Solid Waste and Emergency Response. Municipal Waste Combustion Study: Report to Congress. EPA-530-SW-87-021a. June 1987. p. 86.

6. Memorandum. R.B. Lucas, U.S. EPA, Chemicals and Petroleum Branch, to J. Padgett, U.S. EPA, OAQPS. New Study on the Air Toxics Problem in the United States - POTW Emissions. July 29, 1988. 3 pages.

7. Peters, W.D., U.S. EPA, Pollutant Assessment Branch. Coal and Oil Combustion. July 25, 1988. 6 pages.

8. Memorandum. W.D. Peters, U.S. EPA, PAB, and S.W. Clark, U.S. EPA, STB to R.G. Kellam, Program Analysis and Technology Section, and A.H. Perler, Science and Technology Branch. Risks Associated With Air Emissions from Aeration of Drinking Water. November 10, 1985.

9. U.S. EPA, ESED. National Emission Standards for Asbestos - Background Information for Proposed Standards. Draft. March 5, 1987.

10. South Coast study, p. vi-6.

11. Southeast Chicago study, p. 43.

12. IEMP-Baltimore study, Tables V-8 and V-14.

13. IEMP-Santa Clara study, pp. 3-82 and 3-112.

14. IEMP-Kanawha Valley study, p. 4-94.

15. IEMP-Philadelphia study, p. VI-22.

16. Ambient Air Quality study, Tables 9 and 10.

17. ATERIS Database printouts. 1989.

18. Table E-1. Major Lifetime Risk and Cancer Incidence for the Four Major AN Source Categories. (Personal communication from Ila Cote, USEPA, to Ken Meardon, PES).

19. U.S. EPA. National Emission Standards for Hazardous Air Pollutants; Benzene Emissions from Maleic Anhydride Plants, Ethylbenzene/Styrene Plants, Benzene Storage Vessels, Benzene Equipment Leaks, and Coke By-Product Recovery Plants. Proposed rule and notice of public hearing. July 28, 1938. 53 FR 28496.

20. U.S. EPA. Coke Oven Emissions from Wet-Coal Charged By-Product Coke Oven Batteries -- Background Information for Proposed Standards. EPA-450/3-88-028a. April 1987. p. E-30.

21. Memorandum. L.J. Zaragoza, Pollutant Assessment Branch. Hexachlorobenzene Exposure and Risk Assessment. December 11, 1984. Docket No. A-84-39, Item II-B-1.

The NESHAP/ATERIS data base showed maximum individual risks of 1.0 x 10^{-3} or higher for 12 pollutants in at least one location.[3] These risk estimates are associated with individual sources within specified source categories. The EPA re-estimated the maximum individual risk for 182 of the 205 facilities that were identified as having maximum individual risks of 1 x 10^{-3} or higher in the ATERIS data base. This was done by collecting current information on emissions from these facilities under Section 114 of the Clean Air Act. Overall, the new analysis showed that estimates of maximum individual risk increased for 12 facilities and decreased for 170. One reason for the generally lower risk estimates is that some of the companies have taken steps since the previous emissions data were collected to reduce emissions through process changes and control devices. The new risk estimates generally suggest that the maximum individual risk estimates in the ATERIS data base are too high. Nevertheless, the estimates for some of the 182 facilities analyzed continue to be of serious concern.

On a source category basis, some of the source-specific studies identified a single maximum individual risk value and others reported a range of MIR values. In some instances, only the highest maximum individual risk associated with a specific facility was reported (e.g., POTWs and TSDFs). Other facilities in such source categories would have lower MIRs than those shown in Table 3-2. In other instances, a single MIR was reported that could be expected at a typical, but unspecified facility in a source category (e.g., waste oil combustors). Where a range of MIRs are shown, the values cover either all of the MIRs for each of the facilities modeled (e.g., hazardous waste combustors, drinking water aerators) or the highest MIR expected at typical facilities within the source category (e.g., municipal waste combustors).

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[3] The ATERIS contains data from all stages of air toxics analyses, from the very preliminary to the more detailed. It is not considered an authoritative source for verified estimates of risk attributable to individual point sources, and should not be relied upon as credible estimates of individual source cancer risks. Therefore, the estimates of MIR in the ATERIS data base are subject to significant uncertainty.

Maximum individual risks associated with individual sources within a source category can vary. Table 3-3 illustrates this variation using the distribution of MIR values associated with hazardous waste combustors. Under current conditions (referred to as "baseline" in the table), the majority of these hazardous waste combustors have MIR values of less than 10^{-7}; however, several may have MIR values of 10^{-4}, depending on the type of waste being burned. After compliance with proposed regulations, the highest MIR value decreases to 10^{-5}, and there is a reduction in the number of hazardous waste combustors associated with each level of MIR risk. A second illustration is provided in Table 3-4, which shows the distribution of MIRs associated with coke ovens.

Another way to examine MIR is to look at the number of people exposed to various MIRs. This is illustrated in Table 3-5, which shows the distribution of people exposed to MIRs for drinking water aerators.

Areawide lifetime individual risks are shown in Table 3-6. These risks are in the 10^{-5} to 10^{-4} range. Compared to the maximum individual risks for the corresponding cities shown in Table 3-2, the areawide risks are approximately one order of magnitude lower.

Lifetime individual risks were calculated in the Ambient Air Quality study at monitoring sites in four cities (see Table 3-7). Lifetime individual risks in the Ambient Air Quality study were defined as "the sum of lifetime individual risks for metals, BaP, VOC, and PIC at a monitoring site within a city where a battery of air toxic

TABLE 3-3

DISTRIBUTION OF MAXIMUM LIFETIME INDIVIDUAL CANCER RISKS TO THE MOST EXPOSED INDIVIDUAL FROM HAZARDOUS WASTE COMBUSTORS - BOILERS AND FURNACES

MAXIMUM INDIVIDUAL RISK	TYPE OF WASTE	CONTROL DEVICE PERFORMANCE			
		Base Case[a]		Pessimistic[b]	
		Baseline	After Compliance	Baseline	After Compliance
>1 x 10^{-4}	Base Case	0	0	0	0
1 x 10^{-4}		0	0	0	0
1 x 10^{-5}		10	6	10	6
1 x 10^{-6}		61	48	65	48
1 x 10^{-7}		103	56	101	72
<1 x 10^{-7}		778	650	777	634
Total		952	795[c]	952	759[c]
>1 x 10^{-4}	High Risk	0	0	0	0
1 x 10^{-4}		19	0	21	0
1 x 10^{-5}		100	73	102	73
1 x 10^{-6}		167	52	167	58
1 x 10^{-7}		198	35	207	36
<1 x 10^{-7}		468	595	456	585
Total		952	755[c]	953	752[c]

NOTE: Numbers in table indicate the number of hazardous waste combustors associated with each maximum individual risk level.

SOURCE: U.S. EPA, Office of Solid Waste. Regulatory Impact Analysis for Hazardous Waste Boilers and Industrial Furnaces. Exhibits 7-6, 7-9, 7-12, and 7-14.

[a] "Base case" assumes "typical" removal efficiencies for control devices.

[b] "Pessimistic" assumes removal efficiencies of control devices for toxic metals and hydrogen chloride are several percentages points lower than in the base case in most cases. For organic compounds the difference is several fractions of a percent in most instances.

[c] Difference in total device due to some devices that discontinue burning due to the regulations.

TABLE 3-4

MAXIMUM LIFETIME INDIVIDUAL CANCER RISKS FROM COKE OVEN EMISSIONS

MAXIMUM LIFETIME INDIVIDUAL RISK	NUMBER OF COKE OVENS
$\geq 10^{-2}$	13
10^{-3} to 10^{-2}	25
10^{-4} to 10^{-3}	5

SOURCE: Appendix E. Coke Oven Emissions Risk Assessment for Wet-Coal Charged Coke Oven Batteries.

TABLE 3-5

DISTRIBUTION OF MAXIMUM INDIVIDUAL CANCER RISK AT 22 DRINKING WATER AERATORS

MAXIMUM INDIVIDUAL RISK	NUMBER OF FACILITIES	NUMBER OF PEOPLE EXPOSED
1.9×10^{-5}	1	439
1.3×10^{-5}	1	7
9.5×10^{-6}	1	4
4.6×10^{-6}	1	28
2.9×10^{-6}	1	33
1.8×10^{-6}	1	30
1.4×10^{-6}	1	2
1.1×10^{-6}	1	1
1.0×10^{-6}	1	11
10^{-7}	11	208
10^{-8}	2	13

SOURCE: Memorandum. W.D. Peters, US EPA, Pollutant Assessment Branch, and S.W. Clark, US EPA, Science and Technology Branch, to R.G. Kellam, US EPA, Pollutant Assessment Branch, and A.H. Perler, US EPA, Science and Technology Branch. Risks Associated with Air Emissions from Aeration of Drinking Water. November 13, 1985. Table 5.

TABLE 3-6

AREAWIDE LIFETIME INDIVIDUAL CANCER RISKS FOR SELECTED CITIES

CITY/LOCALE	AREAWIDE LIFETIME INDIVIDUAL RISK	NUMBER OF POLLUTANTS	MAJOR POLLUTANT CONTRIBUTORS TO INDIVIDUAL RISK[a]
Philadelphia[b]	4.0x10^{-5}, 1.2x10^{-4}	7	Benzene, carbon tet. 1,2 dichloropropane
Santa Clara[c]	4x10^{-5}	--	Carbon tetrachloride
Southeast Chicago[d]	2.2x10^{-4}	30	Coke oven emissions, Cr+6
City A[e]	1.4x10^{-4}	20	Formaldehyde, PIC, 1,3-butadiene
City B[e]	4.3x10^{-4}	20	1,3-butadiene, PIC, Cr+6, formaldehyde
City C[e]	2.0x10^{-4}	20	1,3-butadiene, formaldehyde
City D[e]	7.0x10^{-4}	20	PIC, 1-3-butadiene, Cr+6
City E[e]	2.7x10^{-4}	20	PIC, formaldehyde, Cr+6
Kanawha Valley[f]	5.0x10^{-4}, 1.2x10^{-3}	18	Ethylene oxide, 1,3-butadiene
Baltimore[g]	1.3x10^{-4}, 3.3x10^{-4}	9	Benzene, Cr+6
South Coast[h]	1.2x10^{-4}, 2.1x10^{-4}	12	Benzene, Cr+6

Note: In some instances, the areawide lifetime individual risk was calculated by dividing total lifetime cancer cases by exposed population. Where possible and as appropriate, these estimates were adjusted based on unit risk factors used in this study.

[a] Cr+6 = hexavalent chromium
Carbon tet. = carbon tetrachloride

[b] IEMP Philadelphia study, p. V-27. Lower estimated based on modeled data; higher estimate, on monitored data.

[c] IEMP Santa Clara study, p. 3-80.

[d] Southeast Chicago study, p. 38.

[e] Five City study, p. 53.

[f] IEMP Kanawha Valley study, pp. 4-116 and 4-117. Higher estimate based on box model concentrations; the lower, on Gaussian model analysis. These estimates are for the entire Kanawha Valley study area.

[g] IEMP Baltimore study, Tables V-7 and V-13. Range created by range of estimated risk for hexavalent chromium and cadmium.

[h] South Coast study, p. VI-11. Lower estimate based on modeled data; higher estimate on ambient measured data.

TABLE 3-7

SUMMARY OF LIFETIME INDIVIDUAL CANCER RISKS FOR SELECTED CITIES

CITY	LIFETIME INDIVIDUAL RISK[a]	NUMBER OF POLLUTANTS[b]	MAJOR POLLUTANTS CONTRIBUTING TO LIFETIME INDIVIDUAL RISK
Los Angeles	6.6×10^{-4}	17	Formaldehyde, PIC
Baton Rouge	3.8×10^{-4}	16	Ethylene Dichloride, PIC
Boston	3.0×10^{-4}	11	PIC, Chromium (hexavalent)
Chicago	3.2×10^{-3}	14	PIC, Formaldehyde

SOURCE: Ambient Air Quality Study, Table 8.

[a] These risks are the sum of the lifetime individual risks for a number of pollutants using the estimated annual average concentration at a monitoring site within each of the four cities.

[b] Includes nickel, but no cancer incidence was attributed to nickel.

pollutants is being monitored." As seen in Table 3-7, lifetime individual risks on the order of 10^{-4} and higher were found. The magnitude of lifetime individual risks is affected by the number of pollutants as well as the particular pollutants included. The number of pollutants monitored ranged from 11 in Boston up to 17 in Los Angeles. None of the cities had data on 1,3-butadiene, a pollutant found in this study to be one of the major contributors to risk. In addition, formaldehyde data, another major contributor to risk, were unavailable for Baton Rouge and Boston.

Nature of the Cancer Risk Problem

The nature of the cancer risk problem is examined by looking at the relative contributions of individual pollutants and source categories to total estimated nationwide annual cancer incidence. In addition, the geographic variability of the cancer risk is examined by comparing reported ambient concentrations of selected pollutants, estimated annual cancer incidences, and estimated lifetime individual risks.

Individual Pollutants

Table 3-8 presents the percent contribution of individual pollutants to the total estimated cancer cases. The percent contributions were calculated using the point estimates presented in Table 3-1. Where a range is indicated in Table 3-1, the midpoint was used to estimate the pollutant's potential relative contribution. Figure 3-1 illustrates the results presented in Table 3-8.

Based on the estimates in Table 3-1, five pollutants -- PIC, 1,3-butadiene, chromium, benzene, and formaldehyde -- account for approximately 70 percent of the total estimated annual cancer cases. The reader is reminded that there is considerable uncertainty associated

TABLE 3-8

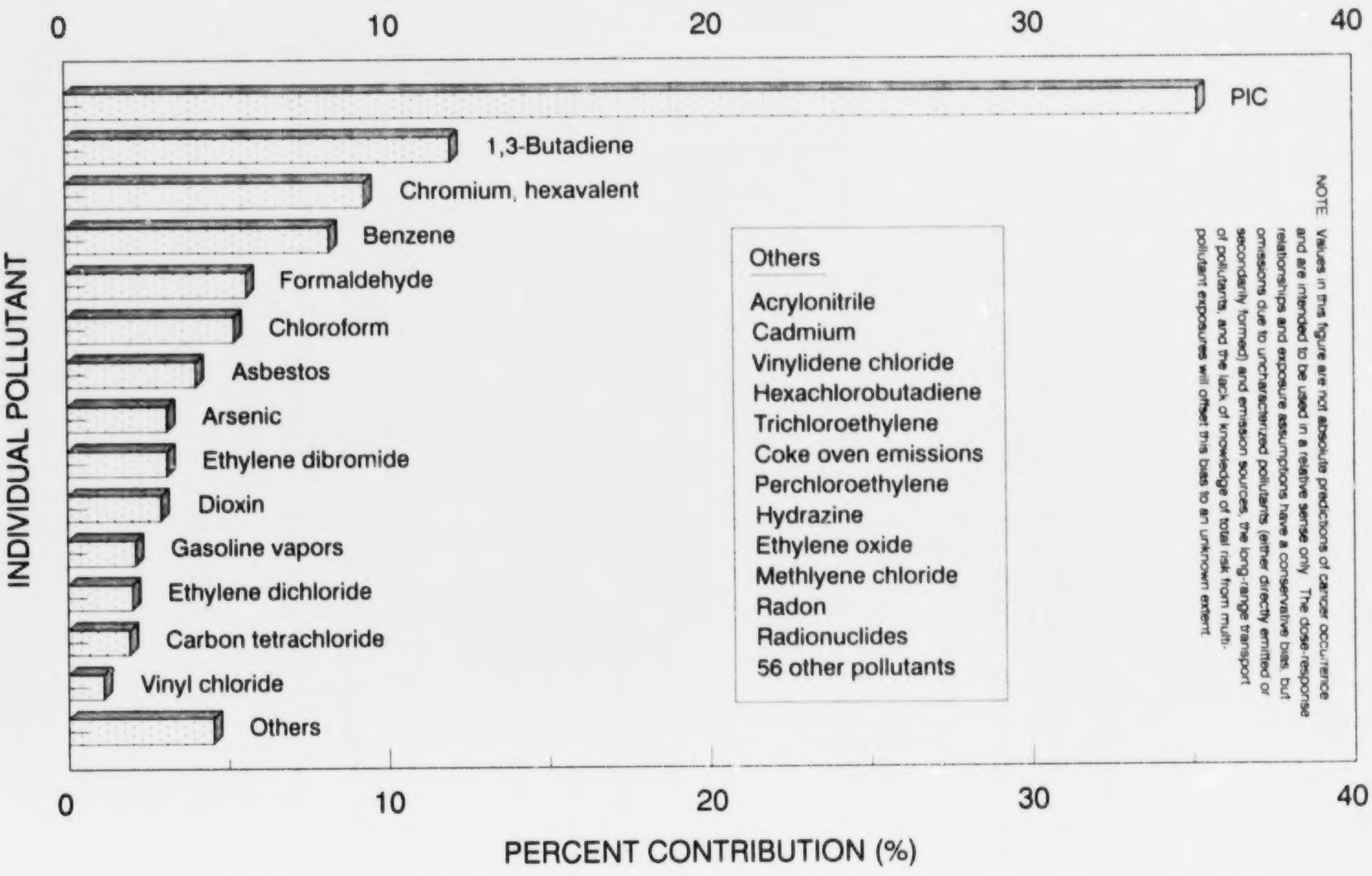
RELATIVE CONTRIBUTION OF INDIVIDUAL POLLUTANTS TO TOTAL ESTIMATED CANCER CASES

POLLUTANT	PERCENT CONTRIBUTION
1. PIC	35.2
2. 1,3-Butadiene	12.0
3. Chromium (hexavalent)	9.3
4. Benzene	8.2
5. Formaldehyde	5.6
6. Chloroform	5.2
7. Asbestos	4.0
8. Arsenic	3.1
9. Ethylene dibromide	3.1
10. Dioxin	2.9
11. Gasoline vapors	2.1
12. Ethylene dichloride	2.0
13. Carbon tetrachloride	1.9
14. Vinyl chloride	1.1
15. Acrylonitrile	0.6
16. Cadmium	0.5
17. Vinylidene chloride	0.5
18. Hexachlorobutadiene	0.4
19. Trichloroethylene	0.3
20. Coke Oven Emissions	0.3
21. Perchloroethylene	0.3
22. Hydrazine	0.3
23. Ethylene oxide	0.3
24. Methylene chloride	0.2
25. Radionuclides[a]	0.1
26. Radon[a]	0.1
27. Miscellaneous	0.7
Totals	100.0

NOTE 1: Values in this figure are not absolute predictions of cancer occurrence and are intended to be used in a relative sense only. The dose-response relationships and exposure assumptions have a conservative bias, but omissions due to uncharacterized pollutants (either directly emitted or secondarily formed) and emission sources, the long-range transport of pollutants, and the lack of knowledge of total risk from multi-pollutant exposures will offset this bias to an unknown extent.

NOTE 2: Total does not sum to 100 percent due to rounding.

[a] Outdoor exposure only; does not include indoor exposure.



NOTE: Values in this figure are not absolute predictions of cancer occurrence and are intended to be used in a relative sense only. The dose-response relationships and exposure assumptions have a conservative bias, but omissions due to uncharacterized pollutants (either directly emitted or secondarily formed) and emission sources, the long-range transport of pollutants, and the lack of knowledge of total risk from multi-pollutant exposures will offset this bias to an unknown extent.

Figure 3-1. Relative Contribution to Total Estimated Nationwide Cancer Cases Per Year, by Pollutant

with the absolute risk estimates for many of the pollutants examined in this study. The relative contribution of any one pollutant is subject to change due to the uncertainties associated with the risk estimates. In addition, the relative contribution may change as new information on as yet unquantified air toxics is obtained.

Source Categories

As noted earlier, over 60 source categories were identified in the studies and reports used. Table 3-9 summarizes the estimated annual cancer cases associated with 40 specified-types of source categories, one aggregate category, and two unspecified categories. The relative contributions of the individual source categories are illustrated in Figure 3-2. It is important to remember that not all source categories that emit air toxics were covered in the studies and reports used for this study. Thus, the relative contributions presented in this study can only reflect the source categories that were covered. As new information is developed, these relative contributions could change, perhaps significantly.

The estimates presented in Table 3-9 reflect the range estimates for modeled estimates only. (By their nature, ambient-measured data do not distinguish between sources.) Although secondary formaldehyde is not a modeled source category per se, two studies (the 5-City study and the Southeast Chicago study) attributed the difference between ambient-measured concentrations and the modeled concentrations to the secondary formation of formaldehyde. It is these results that are included in Table 3-9. Based on the range estimate for the modeled estimates, between 1,430 and 2,538 cancer cases per year are estimated.

Individual source categories have frequently been grouped in two ways: (1) mobile vs. stationary and (2) point vs. area sources. Mobile

TABLE 3-9

SUMMARY OF ESTIMATED CANCER CASES BASED ON MODELED AMBIENT CONCENTRATIONS, BY SOURCE CATEGORY

SOURCE CATEGORY	ANNUAL CANCER CASES	(percent of total)	PRINCIPAL POLLUTANTS
1. Motor Vehicles	769-1,461	(54-58)	PIC, 1,3-butadiene
2. Secondary Formaldehyde	106-154	(7.4-6.1)	Formaldehyde
3. Electroplating	120	(8.4-4.7)	Hexavalent Chromium
4. TSDFs	49-140	(3.4-5.5)	Dioxin
5. Woodsmoke	89	(6.2-3.5)	PIC
6. Asbestos, Demolition	81	(5.6-3.2)	Asbestos
7. Unspecified (point)	27-92	(1.9-3.6)	Arsenic, formaldehyde
8. Cooling Towers	0.01-111	(0.0-4.4)	Hexavalent Chromium
9. Gasoline Marketing	24-75	(1.7-3.0)	Gasoline Vapors, Benzene
10. Solvent Use/Degreasing	22-36	(1.5-1.4)	Perchloroethylene, Methylene Chloride
11. Unspecified (area)	21	(1.5-0.8)	Carbon tetrachloride
12. PVC/EDC/Vinyl Chloride	19	(1.3-0.7)	Vinyl chloride
13. Iron and Steel	17-18	(1.2-0.7)	Coke Oven Emissions, Benzene, PIC
14. Sewage Sludge Incinerators	13	(0.9-0.5)	Cadmium, Vinyl Chloride
15. Municipal Waste Combustors	2-22	(0.1-0.9)	Dioxin
16. Petroleum Refineries	8-14	(0.6-0.6)	Gasoline Vapors, Formaldehyde
17. 1,3-Butadiene Production	10	(0.7-0.4)	1,3-butadiene
18. Styrene-butadiene Rubber Production	10	(0.7-0.4)	1,3-butadiene
19. Coal and Oil Combustion	8-10	(0.6-0.4)	Arsenic
20. POTWs	6	(0.4-0.2)	Vinyl chloride
21. Smelters	3-4	(0.2-0.1)	Formaldehyde
22. Commercial Sterilization/ Hospitals	3-4	(0.2-0.2)	Ethylene Oxide
23. Pesticide Production/Usage	3.4	(0.2-0.1)	Benzene
24. Drycleaning	3	(0.2-0.1)	Perchloroethylene
25. Pulp and Paper Manufacturing	2.1	(0.1-0.08)	Chloroform
26. Chlorinated Drinking Water	1.7	(0.1-0.08)	Chloroform
27. Ethylene Dibromide Production	1.5	(0.1-0.06)	Ethylene Dibromide
28. Polybutadiene Production	1.2	(0.08-0.05)	1,3-butadiene
29. Ethylene Oxide Production	1.2	(0.08-0.05)	Ethylene Oxide
30. Ethylene Dichloride Production	0.8	(0.08-0.05)	Ethylene Dichloride,
31. Waste Oil Burning	0.6	(0.04-0.02)	Arsenic
32. Asbestos Manufacturing	0.5	(0.04-0.02)	Asbestos
33. Asbestos Renovation	0.4	(0.03-0.02)	Asbestos
34. Glass Manufacturing	0.4	(0.03-0.02)	Arsenic
35. Hazardous Waste Combustors	0.3	(0.02-0.01)	Hexavalent Chromium
36. Paint Stripping	0.22	(0.02-0.01)	Methylene chloride
37. Pharmaceutical Manufacturing	0.2-0.4	(0.01-0.02)	Chloroform

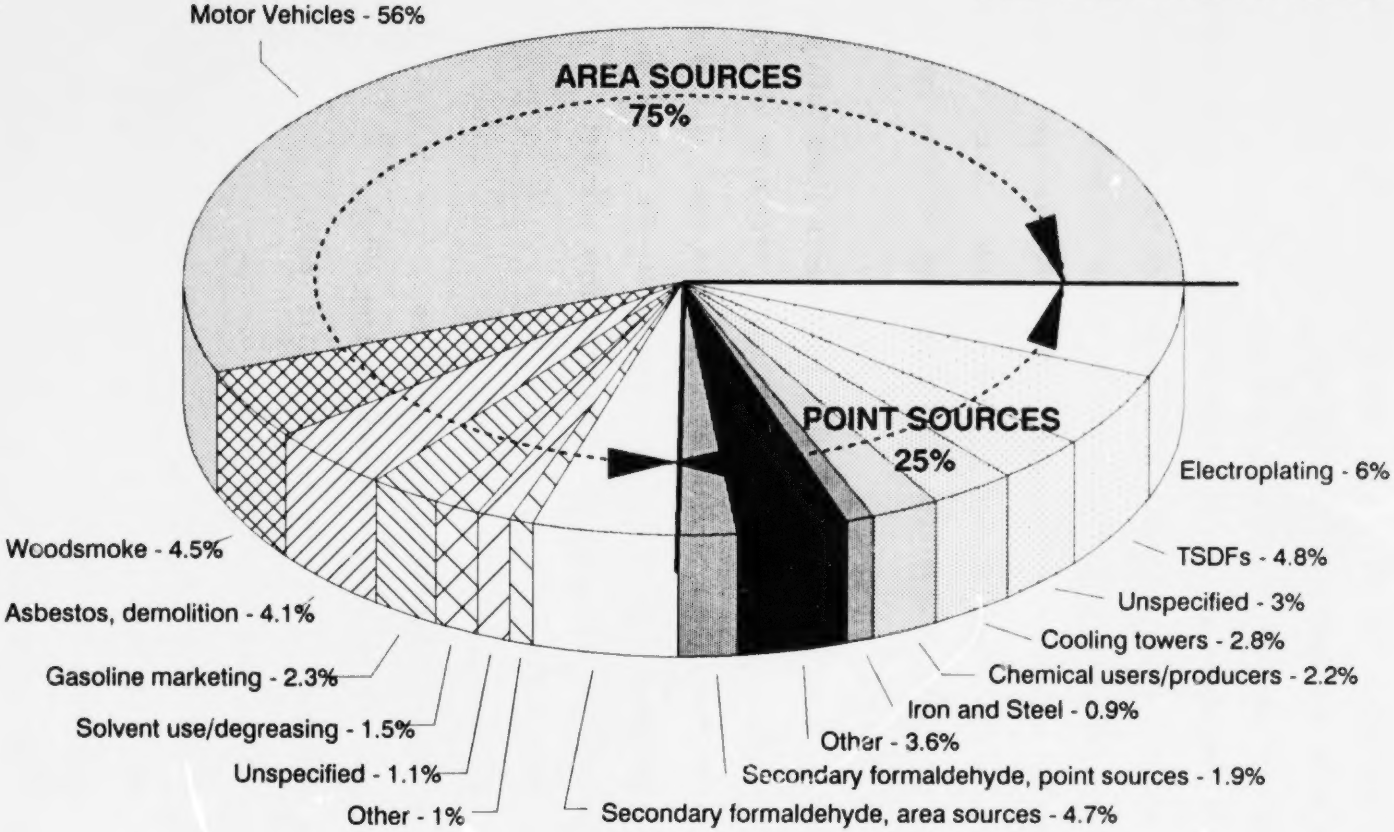
TABLE 3-9

SUMMARY OF ESTIMATED CANCER CASES BASED ON MODELED AMBIENT CONCENTRATIONS, BY SOURCE CATEGORY (concluded)

SOURCE CATEGORY	ANNUAL CANCER CASES	(percent of total)	PRINCIPAL POLLUTANTS
38. Benzene Fugitives	0.2	(0.01-0.01)	Benzene
39. Nitrile Elastomer Production	0.16	(<0.01)	Acrylonitrile
40. ABS/SAN Production	0.13	(<0.01)	Acrylonitrile
41. Asbestos Fabrication	0.13	(<0.01)	Asbestos
42. Benzene Storage	0.1	(<0.01)	Benzene
43. Other	6-13	(0.4-0.5)	Hexavalent Chromium, radon
Total	1,430-2,538		

NOTE: Values in this figure are not absolute predictions of cancer occurrence and are intended to be used in a relative sense only. The dose-response relationships and exposure assumptions have a conservative bias, but omissions due to uncharacterized pollutants (either directly emitted or secondarily formed) and emission sources, the long-range transport of pollutants, and the lack of knowledge of total risk from multi-pollutant exposures will offset this bias to an unknown extent.

[a] Estimated incidences is approximately equally divided between point and area sources.



NOTE Values in this figure are not absolute predictions of cancer occurrence and are intended to be used in a relative sense only. The dose-response relationships and exposure assumptions have a conservative bias, but omissions due to uncharacterized pollutants (either directly emitted or secondarily formed) and emission sources, the long-range transport of pollutants, and the lack of knowledge of total risk from multi-pollutant exposures will offset this bias to an unknown extent.

Figure 3-2. Relative Contribution by Source Categories to Total Estimated Nationwide Cancer Cases Per Year

sources are represented by the "motor vehicle" source category in Table 3-9. All other source categories, except secondary formaldehyde, make up stationary sources. It is sometimes difficult to distinguish between point and area sources. The primary distinguishing feature is the number of sources in a source category. "Area" sources are generally considered to have too many individual sources to develop source-specific data from which to estimate risk for each source. Point sources, in contrast, are "few" enough in number to be located individually to allow source-specific data to be developed, from which risks can be estimated for each individual source. To estimate risks from sources, EPA has developed model algorithms for area sources and for point sources.

For purposes of this study, source categories have been designated as either an "area" source or a "point" source depending on whether the cancer risks were estimated using an area source model algorithm or a point source model algorithm. Using this basis, the following individual source categories are considered area sources:

- motor vehicles
- woodsmoke
- asbestos, demolition and renovation
- gasoline marketing (service stations only)
- coal and oil combustion (residential only)
- solvent use/degreasing
- drycleaning
- pesticide usage
- chlorinated drinking water
- paint stripping

All other sources (except secondary formaldehyde) are considered to be point sources in this study.

The estimated cancer incidence from secondary formation of formaldehyde for selected source categories is shown in Table 3-10. This apportionment was based on the relative percent contributions calculated for specific source categories in the 5-City study. The cancer incidence from secondary formaldehyde was distributed among the major classifications (i.e., mobile vs. stationary and area vs. point), as discussed below. Approximately one-half of the cancer incidence from the "other" source category was from point sources and one-half from area sources. The specific source categories in Table 3-9, however, do not include the estimated cancer incidence from secondary formaldehyde shown in Table 3-10. Instead, a separate "source category" for secondary formaldehyde is shown.

Examining mobile versus stationary sources, approximately 58 percent of the estimated total annual incidence is estimated to occur from motor vehicles (including cancer risk from secondary formaldehyde). Stationary sources account for approximately 42 percent of the total. Of the major stationary sources, two of the top six -- electroplating and cooling towers -- are related to hexavalent chromium, which accounts for the entire estimated risk from these two source categories. The second largest stationary source category is TSDF, in which dioxin is estimated in the study on TSDFs to contribute 65 percent of the total estimated 140 annual cancer cases.[4] In general, while many stationary source categories emit a number of different pollutants, the majority of risk is attributable to a select few in each source category.

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[4] Because of the great uncertainty associated with the estimate of dioxin emissions from TSDFs, this estimate could be substantially lower.

TABLE 3-10

DISTRIBUTION OF ESTIMATED CANCER CASES FROM SECONDARY FORMALDEHYDE FORMATION AMONG SOURCE CATEGORIES[a]

SOURCE CATEGORY	ANNUAL CANCER CASES IN U.S.	PERCENT CONTRIBUTION
Area Sources		
Motor Vehicles	45	34.8%
Solvent Use	38	28.9%
Gasoline Marketing	10	8.3%
Area Source Subtotals	93	71.9%
Point Sources		
Petroleum refining	8	5.9%
Chemical Manufacturing	5	3.7%
Point Sources Subtotal	13	9.6%
Other	24	18.4%
Total Secondary Formaldehyde	130	100%

[a] Distribution of secondary formaldehyde based on data from the 5-City study.

Table 3-11 presents the results in Table 3-9 on an area versus point source basis. Area sources are found to contribute approximately 75 percent off the total number of annual cancer cases (including those from secondary formaldehyde) with point sources contributing approximately 25 percent of the total. Of the area sources, the major source is mobile sources, contributing 78 percent of the total annual incidence attributed to area sources (including the estimated 45 annual cancer cases attributed to mobile sources from secondary formaldehyde, as shown in Table 3-10). For point sources, the largest category is electroplating, which accounts for almost 25 percent of the total point source-related annual incidence. Although the estimates in Table 3-11 add up to "100 percent of the risk," the reader is reminded that this study does not include risk estimates from all known sources. The relative contributions of the types of sources, therefore, could change as additional data on other sources are obtained.

Geographic Variation

As has been stated, the primary purpose of this study is the estimation of nationwide cancer risks. The various studies used to meet this goal illustrate the variation in exposure to different pollutants and in the resulting cancer risk that exists between geographic areas on a county-to-county and city-to-city basis as well as on an intra-city or intra-region basis. Table 3-12 presents ambient-measured concentration data for several selected pollutants and cities. As can be seen in this table, the variation in ambient concentrations depends on the pollutant considered. For example, for the selected cities, ambient benzene concentrations differ by less than a factor of 2. Two of the pollutants vary by factors of approximately 4 to 5. For the other two selected pollutants, ambient concentrations vary by factors of approximately 12

TABLE 3-11

CONTRIBUTION OF AREA VS. POINT SOURCES TO NATIONWIDE ANNUAL CANCER CASES

SOURCE TYPE/ INDIVIDUAL SOURCE CATEGORY	ANNUAL CANCER CASES[a]	PERCENT CONTRIBUTION TO...	
		Nationwide Total	Source Type Total
Area Sources			
Mobile Vehicles	1,115	56.2	75.0
Woodsmoke	89	4.5	6.0
Asbestos, demolition	81	4.1	5.4
Gasoline Marketing	46	2.3	3.1
Solvent Use/Degreasing	29	1.5	1.9
Unspecified/Other	21	1.1	1.4
Commercial Sterilization/ Hospital	3.5	0.2	0.2
Pesticide Usage	3	0.2	0.2
Drycleaning	3	0.2	0.2
Chlorinated Drinking Water	2	0.1	0.1
Coal and Oil Combustion (residential only)	2	0.1	0.1
Asbestos, renovation	0.4	0.02	0.03
Paint Stripping	0.3	0.01	0.02
Secondary Formaldehyde	93	4.7	6.2
Subtotal Area Sources	1,487	75.0	100
Point Sources			
Electroplating	120	6.0	24.1
TSDFs	94	4.8	19.0
Unspecified	59	3.0	11.9
Cooling Towers	56	2.8	11.2
Chemical Users and Producers	43	2.2	8.7
Iron and Steel	17	0.9	3.5
Coal and Oil Combustion (non-residential)	8	0.4	1.5
Sewage Sludge Incinerators	13	0.7	2.6
Municipal Waste Combustors	12	0.6	2.4
Petroleum Refineries	11	0.6	2.2
Miscellaneous	11	0.5	2.2
POTWs	6	0.3	1.1
Manufacturing	6	0.3	1.3
Gasoline Marketing	3	0.2	0.6
Secondary Formaldehyde	38	1.9	7.5
Subtotal Point Sources	497	25.0	100
TOTAL - All sources	1,984	100	

[a] Based on mid-point of estimate from Table 3-9.

TABLE 3-12

COMPARISON OF MEASURED AMBIENT CONCENTRATIONS OF SELECTED POLLUTANTS IN SELECTED CITIES

POLLUTANT	CITY						
	A	B	C	D	E	F	G
Benzene	8.2	7.9	8.4	10.8	9.8	13.10	8.8
Chloroform	1.0	6.2	4.6	4.6	17.4	16.6	1.2
Ethylene dibromide	0.06	0.05	0.2	0.04	0.07	-	-
Methylene chloride	6.1	3.7	2.1	5.2	7.6	-	-
Perchloroethylene	1.5	3.6	5.9	4.5	2.4	5.8	0.5

NOTE: All numbers are in terms of μg/m3.

SOURCE: Ambient Air Quality Study, data worksheets.

Key: A = Bakersfield, CA
B = Newark, NJ
C = Philadelphia, PA
D = Elizabeth, NJ
E = Camden, NJ
F = Baltimore, MD
G = Baton Rouge, LA

for perchloroethylene and 17 for chloroform. The degree of variation presented in Table 3-12 depends upon the pollutants and cities selected. Nevertheless, the point is still the same regardless of which pollutants or cities are selected -- ambient concentrations vary between cities.

Ambient concentrations can also vary within a city or within a specified geographic locale (e.g., the South Coast Air Basin, the Kanawha Valley). Table 3-13 presents ambient-measured concentrations for selected pollutants at different locations in Baltimore, the South Coast Air Basin, and the Kanawha Valley. In general, the variation in ambient concentrations within each area is approximately the same as the variation in Table 3-12 for comparable pollutants.

As might be expected, the variations in ambient concentrations for pollutants can lead to variations in the number of cancer cases between geographic areas and in the estimates of cancer cases per year per million population. Results from eight studies are presented in Table 3-14. As seen in this table, annual cancer cases varied from a low of 0.03 per year to a high of 128 per year. This reflects a combination of

TABLE 3-13

INTRACITY COMPARISON OF AMBIENT CONCENTRATIONS ($\mu g/m^3$) FOR SELECTED POLLUTANTS IN THREE CITIES, BY LOCATION

POLLUTANT	BALTIMORE[a]										SOUTH COAST AIR BASIN[b]					KANAWHA VALLEY[c]				
	1	2	3	4	5	6	7	8	9	10	1	2	3	4	5	1	2	3	4	5
Benzene	12	9.5	10.2	12.9	12.6	5.5	12.1	10.6	7.8	10.3	4.9	4.1	4.2	2.5	3.4	1.3	1.9	3	8.6	2.9
Carbon tetrachloride[d]	1.1	0.9	0.6	1.3	0.9	0.9	1.2	1.4	0.7	0.6	0.095	0.1	0.11	0.099	0.12	0.5	ND	1.37	0.5	1.6
Chloroform	2.1	0.4	0.2	0.7	1	3.2	2.6	4.7	0.6	1.1	0.063	0.082	0.11	0.053	0.071	ND	ND	0.5	8.6	4.4
Perchloroethylene	4.8	5.4	7	6	9.3	1.5	3.2	3.9	2.9	2.4	1.6	1	1.2	0.45	0.7	ND	ND	ND	46.3	ND
Trichloroethylene	0.9	0.5	0.5	3.9	1.4	0.4	1.1	1	0.2	0.3	0.4	0.29	0.34	0.1	0.37	-	-	-	-	-
1,1,1-trichloroethane	-	-	-	-	-	-	-	-	-	-	7.1	3	2.4	1.1	1.6	-	-	-	-	-

NOTE: ND = not detected.

[a] IEMP - Baltimore study, Table v-3.

[b] South Coast study, pp. V-4 and V-5.

[c] IEMP - Kanawha Valley study, Chapter 4, Tables 16 through 19.

[d] Background levels of carbon tetrachloride have been established in several comprehensive studies as being around 0.8 $\mu g/m^3$. In addition, carbon tetrachloride is difficult to measure. Thus, concentrations much below this level, as seen in the South Coast Air Basin data, must be viewed at this time as resulting from test error.

TABLE 3-14

VARIATION IN ANNUAL CANCER CASES AND CANCER RATES DUE TO EXPOSURE TO OUTDOOR AIR TOXICS BY GEOGRAPHIC LOCALES

GEOGRAPHIC LOCALE	ESTIMATED ANNUAL CANCER CASES	CANCER CASES PER YEAR PER MILLION POPULATION[b]	STUDY
Baltimore	2.95-7.15	1.9-4.7	IEMP-Baltimore
Kanawha Valley	1.77	17.8	IEMP-Kanawha Valley
Philadelphia	0.42	0.26	IEMP-Philadelphia
Santa Clara	1.85	1.4	IEMP-Santa Clara
City A	3.1	2.1	5 City
City B	67.7	6.2	5 City
City C	1.1	2.8	5 City
City D	14.1	10.0	5 City
City E	6.7	3.9	5 City
Southeast Chicago	1.26	3.2	Southeast Chicago
South Coast	19-33	1.7-3.0	South Coast
County A	128.3	17.2	35-County[a]
B	38.4	20.4	
C	24.1	12.5	
D	22.8	38.3	
E	14.6	11.3	
F	12.4	14.6	
G	7.8	9.2	
H	7.6	9.7	
I	6.6	8.7	
J	5.5	3.5	
K	4.8	19.2	
L	3.1	2.8	
M	1.6	1.9	
N	1.1	3.0	
O	0.6	4.2	
P	0.2	1.2	
Q	0.1	0.6	
R	0.03	1.6	

[a] Selected counties.

[b] Derived by dividing estimated annual cancer cases by the population in the geographic locale.

different exposure levels and the size population exposed to those levels. Cancer rates varied from a low of 0.26 to a high of 38.3 cancer cases per year per million population. These are equivalent to areawide lifetime individual risks of approximately 2 x 10^{-5} to 3 x 10^{-3} for the exposed populations. In general, a lower absolute number of cancer cases corresponded to a lower cancer rate. A notable exception is County K from the 35-County study, where a "modest" number of estimated annual cancer cases (4.8) had one of the highest cancer rates (19.2 cancer cases per year per million population).

Variation in lifetime individual risk between and within cities can also be examined. Areawide lifetime individual risks for selected cities were presented earlier in Tables 3-6 and 3-7. The lifetime individual risks among the cities/locales shown in these two tables, however, were essentially on the same order of magnitude with one another (approximately 10^{-4}). The areas shown in Tables 3-6 and 3-7 are urban areas, with the exception of the Kanawha Valley, which is classified primarily as rural. The Kanawha Valley, on the other hand, is a fairly highly industrialized area. Because of the large number of factors that differed in deriving these risk estimates, it is difficult, if not impossible, to say why such a relatively narrow range is observed. The small range may point to a relatively consistent areawide lifetime individual risk regardless of the urban area or industrialized area in which one lives.

Table 3-15 presents maximum individual risks associated with various cities or specific geographic locales. The maximum individual risks in Table 3-15, however, are not necessarily directly comparable to each other, because they vary in manner in which they were estimated and

TABLE 3-15

VARIATION IN MAXIMUM LIFETIME INDIVIDUAL RISK, BY LOCATION

LOCATION	MAXIMUM LIFETIME INDIVIDUAL RISKS	COMMENT	REFERENCE
South Coast[b]	10^{-3}	MEI[a] to hexavalent chromium	1
Southeast Chicago[c]	1×10^{-3}	MEI, additive, 30 pollutants	2
Baltimore	1×10^{-4}	MEI to benzene and to chloroform	3
Philadelphia[d]	7×10^{-6} to 3×10^{-4}	MEI, additive, 9 pollutants	4
Santa Clara	3×10^{-4}	MEI to emissions at a traffic intersection	5
Kanawha Valley[e]			6
Belle	3×10^{-3}	additive, 3 pollutants	
Charleston	6×10^{-3}	additive, 4 pollutants	
Institute	8×10^{-3}	additive, 6 pollutants	
Nitro	8×10^{-6}	additive, 9 pollutants	

NOTE: Values have been adjusted to reflect unit risk factors used in this study wherever possible.

[a] MEI = maximum exposed individual

[b] Maximum exposed individual risk based on model-predicted exposures in the South Coast study rather than the monitored exposures in the study.

[c] 8×10^{-4} of additive lifetime risk was attributed to five pollutants from steel mills. Southeast Chicago study, p. 43.

[d] MEI risks were calculated for eight locations in the city. The range reflects the high and low MEI risks estimated.

[e] The individual risks calculated in the IEMP-Kanawha Valley Study were for neighborhood sites with suspected highest exposures from point source pollutants.

REFERENCES TO TABLE 3-15

1. South Coast study, p. VI-6.
2. Southeast Chicago study, p. x.
3. IEMP - Baltimore study, Table V-8. Based on exposure at the site of maximum average concentration.
4. IEMP - Philadelphia study, p. VI-49.
5. IEMP - Santa Clara study, p. 3-82.
6. IEMP - Kanawha Valley study, pp. 4-73, 4-84, 4-95, and 4-99.

the number of pollutants included in the risk estimate. For the locations shown, maximum individual risks are in the neighborhood of 10^{-3} and 10^{-4}. The results from the IEMP-Philadelphia study illustrate that the MEI level can vary within subareas of a city by a factor of 40 (7×10^{-6} to 3×10^{-4}), while the IEMP-Kanawha Valley study's results show a 1,000-fold difference in maximum individual risk for one of the locales in the Kanawha Valley versus the other three locales (10^{-6} vs. 10^{-3}). Table 3-16 shows additional details on the variation in maximum individual risk within the Kanawha Valley.

The pollutants that are the most important contributors to annual incidence and the source categories that emit those pollutants and to the individual risk in a geographic area can also vary from one area to another. Table 3-17 illustrates some of the annual incidence variation for five pollutants and the source categories that emit two of these pollutants across six selected cities. For example, among the individual pollutants, benzene is estimated to contribute a relatively consistent percentage, between approximately 5 and 10 percent of total cancer cases for the six cities. In contrast, 1,3-butadiene is seen in Table 3-17 to contribute a much wider range, between 6 and 48, percent of total cancer cases across the five cities. Among source categories, road vehicles are consistently a major contributor to annual incidence attributed to benzene in each city, contributing between 45 and 81 percent of the total cancer cases attributed to benzene, and are the most important source category of benzene-related incidence in five of the six cities. In contrast, the relative contribution of "iron and steel" to benzene incidence varies dramatically between cities, ranging from 0 percent in four of the six cities to 25 percent in City D and over 50 percent in Southeast Chicago. Along the same lines, 100 percent

TABLE 3-16

ESTIMATES OF MAXIMUM LIFETIME INDIVIDUAL CANCER RISKS IN NEIGHBORHOODS SURROUNDING FACILITIES IN THE KANAWHA VALLEY

TYPE OF INDIVIDUAL RISK	LOCALE			
	Belle	Charleston/ South Charleston	Institute	Nitro
Highest (number of people exposed)	2.8×10^{-3} (600)	6×10^{-3} (2,700)	8×10^{-3} (1,300)	8×10^{-6} (1,453)
Range of Maximum Risks in Remaining Neighborhoods	7×10^{-5} to 4×10^{-4}	2×10^{-4} to 3×10^{-3}	2×10^{-4} to 2×10^{-3}	9×10^{-7} to 6×10^{-6}
Average Risk	2.2×10^{-4}	2.8×10^{-4}	1.1×10^{-3}	3.2×10^{-6}
Population in locale (number of people)	15,530	51,750	22,390	9,990

NOTE 1: Values have been adjusted to reflect the unit risk factors used in this study.

NOTE 2: These risk estimates are based on pollutants from point sources; risk from area source pollutants are not included.

SOURCE: IEMP-Kanawha Valley Study, pp. 4-73, 4-77, 4-84, 4-88, 4-95, 4-97, 4-102, and 4-106.

TABLE 3-17

CITY-TO-CITY VARIATION IN RELATIVE CONTRIBUTION OF SELECTED POLLUTANTS TO TOTAL ANNUAL CANCER INCIDENCE

ITEM	CITY[a]					
Pollutant (Source Category)	A	B	C	D	E	SOUTHEAST CHICAGO[b]
Benzene	8.8%	10.1%	4.8%	9.6%	7.0%	4.8%
(Road vehicles	73%	81%	67%	56%	63%	45%)
(Gas marketing	7%	3.6%	0	1%	3%	2.3%)
(Iron & Steel/Steel Mills	0	0	0	24%	0	52%)
1,3-Butadiene	19.3%	23.9%	48.4%	16%	13%	6.4%
(Road vehicles	100%	100%	17%	100%	100%	100%)
(Chemical Mfg.	0	0	83%	0	0	0)
Formaldehyde	34.5%	23.0%	24%	6.9%	18.9%	17%
Chromium	5.2%	21.3%	16%	14%	16%	16%
Methylene chloride	2.3%	3.9%	1.6%	2.2%	3%	0.2%

[a] 5-City study. Derived from data worksheets.

[b] Southeast Chicago study, p. 33. Relative contributions have been adjusted based on unit risk factors listed in Table 2-6 in this report.

of the estimated 1,3-butadiene related cancer cases are due to emissions from road vehicles in five of the six cities. For City C, however, over 80 percent of the 1,3-butadiene related cancer cases are attributed to chemical manufacturing and less than 20 percent to road vehicles.

Table 3-18 illustrates the variation in maximum individual risk for individual pollutants. The data are taken from the IEMP-Baltimore study, and show the exposed population to each pollutant as well. The range in "maximum" individual risks is from 10^{-6} to 10^{-4}. In Table 3-19, the individual pollutant contributors to the highest estimated individual risk grid cell in the Southeast Chicago area are presented. As seen in Table 3-19, coke oven emissions contribute over 77 percent of the total individual risk.

Table 3-20 illustrates the areawide lifetime individual risk associated with individual pollutants based upon data from the IEMP-Philadelphia study. Both monitored and modeled results are presented. The range of areawide lifetime individual risks for individual pollutants is from 10^{-6} to 10^{-5}.

Variation in lifetime individual risk across source categories is illustrated in Tables 3-21 through 24. Tables 3-21 and 22 report maximum individual risks for two cities. Specific sources show maximum individual risks in the range of 10^{-7} to 10^{-4}.

Tables 3-23 and 24 show areawide lifetime individual risks for specific sources. Table 3-23 shows area and point sources in Santa Clara, while Table 3-24 shows area and point sources for the Kanawha Valley. In the Santa Clara study, area and point sources are found to be the major contributor to total areawide lifetime individual risk. On the other hand, point sources are found in the Kanawha Valley to be the major contributor to total areawide lifetime individual risks. Both

TABLE 3-18

MAXIMUM LIFETIME INDIVIDUAL CANCER RISKS IN BALTIMORE BY INDIVIDUAL POLLUTANT[a]

POLLUTANT	MAXIMUM LIFETIME INDIVIDUAL RISK	EXPOSED POPULATION[b]
Benzene	1.0×10^{-4}	48,771
Trichloroethylene	6.7×10^{-6}	48,771
Perchloroethylene	5.4×10^{-6}	14,270
Ethylene dichloride	6.8×10^{-5}	12,880
Chloroform	1.1×10^{-4}	23,997
Carbon tetrachloride	2.1×10^{-5}	23,997
1,2-dichloropropane	3.6×10^{-5}	16,848
Chromium (hexavalent)[c]	0 to 3.6×10^{-4}	490,690
Cadmium[d]	0 to 3.6×10^{-6}	118,411

NOTE: Values have been adjusted to reflect the unit risk factors used in this study.

SOURCE: IEMP-Baltimore Study, Tables V-8 and V-14..

[a] Except for cadmium, individual risks were calculated using the maximum observed ambient concentration measured across all monitoring sites. Measured cadmium concentrations were below detection limits. For screening purposes, the Baltimore study calculated risks assuming a range in ambient concentrations from zero to the upper end of the detection limit (about 0.002 $\mu g/m^3$).

[b] The exposed population is in the grid cell at the monitoring site of maximum concentration.

[c] Range indicates possible ambient levels of hexavalent chromium, from 0 percent to 100 percent.

[d] Range indicates possible ambient levels from 0.00 $\mu g/m^3$ to the upper end of the detection limit (about 0.002 $\mu g/m^3$).

TABLE 3-19

RELATIVE CONTRIBUTION OF INDIVIDUAL POLLUTANTS TO MAXIMUM LIFETIME INDIVIDUAL RISK OF CANCER IN THE SOUTHEAST CHICAGO AREA

POLLUTANT	MAXIMUM INDIVIDUAL RISK	% CONTRIBUTION
Coke Oven Emissions[a]	7×10^{-4}	77%
Benzene	6×10^{-5}	7
Chromium	5×10^{-5}	6
Formaldehyde	4×10^{-5}	4
POM	3×10^{-5}	3
Arsenic	2×10^{-5}	2
Cadmium	1×10^{-5}	1
Carbon tetrachloride	1×10^{-5}	1
Others	$<1\times10^{-5}$	<1
TOTAL	9×10^{-4}	100

SOURCE: Southeast Chicago study, p. 43.

[a] "Coke oven emissions" is a mixture of compounds that includes other pollutants such as benzene and POM. The cancer risk estimates are for the full mixture of coke oven emissions.

TABLE 3-20

AREAWIDE LIFETIME INDIVIDUAL RISKS OF CANCER: MONITORED VS. MODELED AMBIENT AIR CONCENTRATIONS IN PHILADELPHIA

POLLUTANT	AREAWIDE LIFETIME INDIVIDUAL RISK	
	MONITORED	MODELED
Chloroform	6.9×10^{-6}	4.6×10^{-6}
Ethylene dichloride	1.0×10^{-5}	1.0×10^{-5}
Carbon tetrachloride	2.7×10^{-5}	1.5×10^{-6}
Benzene	5.0×10^{-5}	1.9×10^{-5}
Trichloroethylene	2.7×10^{-6}	1.7×10^{-6}
1,2-dichloropropane	2.2×10^{-5}	9.0×10^{-6}
Perchloroethylene	2.8×10^{-6}	2.1×10^{-6}
CUMULATIVE	1.2×10^{-4}	4.8×10^{-5}

NOTE: Values have been adjusted to reflect the unit risk factors used in this study.

SOURCE: IEMP-Philadelphia, p. V-27.

TABLE 3-21

ESTIMATES OF MULTI-POLLUTANT LIFETIME CANCER RISKS TO THE MOST EXPOSED INDIVIDUAL TO VARIOUS SOURCES IN PHILADELPHIA

MEI LOCATION	MAXIMUM INDIVIDUAL RISK	COMMENT
Northeast Water Control Plant	6.2×10^{-5}	8 pollutants
Refinery B	1.4×10^{-5}	3 pollutants
Chemical Manufacturer	2.3×10^{-4}	3 pollutants
Plastic Cabinet Mfr.	8.2×10^{-7}	1 pollutant
Pharmaceutical Mfr.	3.2×10^{-4}	3 pollutants
Garment Mfr.	1.7×10^{-5}	1 pollutant
Refinery A	3.1×10^{-5}	3 pollutants
Industrial Dry Cleaner	2.8×10^{-5}	1 pollutant

NOTE: Where possible and as needed, the values have been adjusted to reflect the unit risk factors used in this study.

SOURCE: IEMP-Philadelphia Study, p. VI-49.

TABLE 3-22

ESTIMATED CANCER RISK TO MAXIMUM EXPOSED INDIVIDUALS TO ORGANIC GASES IN SANTA CLARA FOR SELECTED SOURCES

SOURCE TYPE	MAXIMUM INDIVIDUAL RISK
Traffic Intersections	3×10^{-4}
Hospitals	2×10^{-4}
Pharmaceutical Manufacturer	1×10^{-4}
Computer Equipment Mfr.	4×10^{-5}
Industrial Facility	3×10^{-5}
Fuel Pipeline	2×10^{-5}
Drycleaners	1×10^{-5}
Sewage Treatment Plants	5×10^{-6}
Gasoline Station Pump	4×10^{-6}
Groundwater Aeration	2×10^{-7}

SOURCE: IEMP-Santa Clara study, p. 3-82.

TABLE 3-23

AREAWIDE LIFETIME INDIVIDUAL RISK OF CANCER FROM LIFETIME EXPOSURE TO ORGANIC GASES IN SANTA CLARA

SOURCE CATEGORY	AREAWIDE INDIVIDUAL RISK
Burning of Waste Material	4×10^{-8}
Combustion of Fuels	1×10^{-6}
Degreasers	8×10^{-7}
Drycleaners	8×10^{-7}
Fuels Distribution	1×10^{-6}
Industrial Solvents Coating	3×10^{-6}
Mobile Sources	1×10^{-5}
Off-Highway Mobile Sources	3×10^{-7}
Other Chem./Indust.	2×10^{-7}
Other Organics Evaporation	4×10^{-7}
Pesticides Usage	8×10^{-7}
Area Source Total	2×10^{-5}
25 Point Sources Total	6×10^{-6}
Carbon Tetrachloride	1×10^{-5}
TOTAL	4×10^{-5}

SOURCE: IEMP-Santa Clara study, p. 3-80.

TABLE 3-24

ESTIMATES OF AREAWIDE LIFETIME INDIVIDUAL RISKS OF CANCER ACROSS AREA AND POINT SOURCES IN THE KANAWHA VALLEY

SOURCE TYPE	LOCALE			
	Belle	Charleston/ South Charleston	Institute	Nitro
Area				
Gasoline Marketing	1.1×10^{-6}	1.4×10^{-6}	7.9×10^{-7}	3.4×10^{-7}
Heating	1.3×10^{-5}	1.3×10^{-5}	1.3×10^{-5}	4.2×10^{-6}
Road Vehicles	3.1×10^{-5}	5.1×10^{-5}	2.6×10^{-5}	9.8×10^{-6}
Solvent Use	7.7×10^{-6}	1.3×10^{-5}	6.3×10^{-6}	1.8×10^{-6}
Waste Oil Burning	6.8×10^{-7}	1.2×10^{-6}	6.5×10^{-7}	2.4×10^{-7}
Area Subtotal	5.3×10^{-5}	8.0×10^{-5}	4.7×10^{-5}	1.6×10^{-5}
Point	2.2×10^{-4}	2.8×10^{-4}	1.1×10^{-3}	3.0×10^{-6}
TOTAL	3.0×10^{-4}	3.6×10^{-4}	1.1×10^{-3}	1.9×10^{-5}
Population in Locale	15,530	51,750	22,390	9,990

SOURCE: IEMP-Kanawha Valley Study. Tables 32, 40, 45, 52, and 54. Values for area sources could not be adjusted using the unit risk factors in this study. However, the net effect is expected to be small. Values for point sources have been adjusted using the unit risk factors in this study.

studies show similar risks from area sources (10^{-5} range). The difference in relative contributions is due to the presence or absence of point sources. The Kanawha Valley is a relatively heavily industrialized area, with significant point sources, whereas the Santa Clara area is much less industrialized. Thus, the relative contributions of point and area sources to total areawide lifetime individual risks is consistent with the character of the two study areas.

The areawide lifetime individual risk data from area sources in the Kanawha Valley Study show relatively consistent percentage contribution among the same source category between locales. Among area-type sources, "mobile sources" as a source category is found to be the largest contributor to areawide lifetime risks in both the Santa Clara and Kanawha Valley studies.

Comparison with the Results from the 1985 Six-Month Study

The results of the present study are compared with the results of the 1985 Six-Month Study. This is done in two ways. First, a comparison of estimated nationwide cancer cases is made to examine the magnitude of the problem. Second, a comparison of the nature of the problem is presented by examining the pollutants and the source categories that appear to be the greatest contributors to risk.

Magnitude of the Problem

Tables 3-25 and 3-26 compare the cancer rates (i.e., annual cancer cases per million population) and annual cancer cases, respectively, estimated for three studies presented in the 1985 Six-Month Study with the point (or range) estimates of this study. As seen in these two tables, the present study's low end estimated total cancer cases per year per million population and the nationwide number of annual cancer

TABLE 3-25

COMPARISON OF ANNUAL CANCER CASES PER MILLION POPULATION WITH 1985 SIX-MONTH STUDY

POLLUTANT	1985 SIX-MONTH STUDY NESHAP	 35 County	 Ambient Air Quality	THIS STUDY
Arsenic	0.02	0.02	0.26	0.28
Benzene	0.14	0.39	1.02	0.75
1,3-butadiene	<0.001	<0.001	--	1.11
Cadmium	0.04	0.02	0.06	0.04
Carbon tetrachloride	0.06	0.004	0.19	0.17
Chloroform	<0.01	0.002	0.07	0.48
Chromium (hexavalent)	1.43	0.29	1.05	0.61-1.1
Dioxin	--	--	--	0.008-0.52
Ethylene Oxide	0.21	N/A	N/A	0.03
Ethylene dibromide	0.12	0.02	N/A	0.28
Ethylene dichloride	<0.01	0.03	0.05	0.19
Formaldehyde	0.01	0.21	0.83	0.52
Gasoline vapors	N/A	0.15	N/A	0.08-0.32
Perchloroethylene	0.01	0.14	0.10	0.03
Trichloroethylene	0.04	0.15	0.08	0.03
Vinyl chloride	0.05	0.17	--	0.10
Vinylidene chloride	<0.01	N/A	0.27	0.04
Other	0.11	0.35	0.01	0.13
Risk Estimates from Other EPA Efforts[a]				
Asbestos	0.50	0.50	0.50	0.37
Gasoline Marketing	0.20	--	0.20	--
PIC	2.65	2.60	2.68	1.83-4.67
Radionuclides[b]	0.07	0.07	0.07	0.02
TOTAL	5.6	4.9	7.4	7.2 - 11.3

NOTE: Values in this figure are not absolute predictions of cancer occurrence and are intended to be used in a relative sense only. The dose-response relationships and exposure assumptions have a conservative bias, but omissions due to uncharacterized pollutants (either directly emitted or secondarily formed) and emission sources, the long-range transport of pollutants, and the lack of knowledge of total risk from multi-pollutant exposures will offset this bias to an unknown extent.

[a] Except for PIC in the 35-County study, these estimates of cancer incidences were not part of the individual results of the NESHAP, 35-County, and Ambient Air Quality studies. The 1985 Six-Month Study included these estimates for these pollutants to provide for a more complete accounting of information available to the 1985 Six-Month Study.

[b] Includes radon.

TABLE 3-26

COMPARISON OF ANNUAL CANCER CASES WITH 1985 SIX-MONTH STUDY

POLLUTANT	1985 SIX-MONTH STUDY NESHAP	35 County	Ambient Air Quality	THIS STUDY
Acrylonitrile	0.42	4.2	--	13
Arsenic	4.7	1.1	60	68
Benzene	32.3	18.5	234	181
1,3 butadiene	0.01	0.01	--	266
Cadmium	8.5	1.1	14.6	10
Carbon tetrachloride	14	0.2	43	41
Chloroform	0.27	0.1	17	115
Chromium (hexavalent)	330	13.4	242	147-265
Coke Oven Emissions	8.6	2.4	--	7
Dioxin	--	--	--	2-125
Ethylene Oxide	47.8	--	--	6
Ethylene dibromide	26.7	1.0	--	68
Ethylene dichloride	0.9	1.5	11	45
Formaldehyde	1.6	10	191.3	124
Gasoline vapors	--	6.8	--	19-76
Methylene chloride	1	--	7.4	5
Perchloroethylene	2.9	6.7	22	6
Trichloroethylene	9.7	6.8	18	7
Vinyl Chloride	11.7	8.2	--	25
Vinylidene chloride	0.04	--	62	10
Other	2.9	0	1	30
Subtotals	504	207	1539	1,195-1,493
Risk Estimates from Other EPA Efforts[a]				
Asbestos	115	23.7	115	88
Gasoline Marketing	46	--	46	(see gas vapor)
PIC	610	125.1	615.4	438-1120
Radionuclides/Radon	16	3.3	16	5
TOTAL	1291	234	1716	1,726-2,706

NOTE: Values in this figure are not absolute predictions of cancer occurrence and are intended to be used in a relative sense only. The dose-response relationships and exposure assumptions have a conservative bias, but omissions due to uncharacterized pollutants (either directly emitted or secondarily formed) and emission sources, the long-range transport of pollutants, and the lack of knowledge of total risk from multi-pollutant exposures will offset this bias to an unknown extent.

[a] Except for PIC in 35-County study, these estimates of concern incidences were not part of the individual results of the NESHAP, 35-County, and Ambient Air Quality studies. The 1985 Six-Month study included these estimates for these pollutants to provide for a more complete accounting of information available to the 1985 Six-Month study.

cases are essentially the same as the original Ambient Air Quality study in 1985 Six-Month Study, which was larger than the other two studies in the 1985 Six-Month Study. The upper end of the estimated cancer rate and annual cancer cases of the present study are approximately 1.5 to 2 times larger than the NESHAP or Ambient Air Quality studies in the 1985 Six-Month Study. Using a 1986 U.S. population of 240 million, the present study estimates up to approximately 500 to 900 more cancer cases per year nationwide than either the NESHAP or Ambient Air Quality study results in the 1985 Six-Month Study.

There are several factors that account or may account for this apparent increase in estimated risk. One factor is that this study includes more pollutants for which risks have been estimated than were included in the 1985 Six-Month Study. Most of these pollutants are the result of the Sewage Sludge Incinerator, Hazardous Waste Combustion, and TSDF studies being available for inclusion. On an individual pollutant basis, the potentially most important addition from TSDFs is dioxin, for which up to 92 annual cancer cases were estimated based on data in the TSDF study. As shown in Table 3-26 by the large range in risk (2 to 125 annual cancer cases), there is substantial uncertainty associated with the risk estimate for dioxin in this study.

A second factor that accounts for an increase in the estimated cancer risk is the changes, some of which are significant, that have occurred to unit risk factors. Table 3-27 compares the unit risk factors used in the 1985 Six-Month Study with those used in this study for those pollutants for which the unit risk factor has changed. As seen in this table, the unit risk factors have changed in a few instances by relatively small amounts (±25 percent). In some instances, cases are essentially the same as the original Ambient Air Quality study in 1985 Six-Month Study, which was larger than the other two studies in the 1985 Six-Month Study. The upper end of the estimated cancer rate and annual cancer cases of the present study are approximately 1.5 to 2 times larger than the NESHAP or Ambient Air Quality studies in the 1985 Six-Month Study. Using a 1986 U.S. population of 240 million, the present study estimates up to approximately 500 to 900 more cancer cases per year nationwide than either the NESHAP or Ambient Air Quality study results in the 1985 Six-Month Study.

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TABLE 3-27

COMPARISON OF UNIT RISK FACTORS

POLLUTANT	MAY 1985	JUNE 1988	% CHANGE
Acrylamide	1.7×10^{-5}	1.1×10^{-3}	+ 6400
Benzene	6.9×10^{-6}	8.3×10^{-6}	+ 20
BaP	3.3×10^{-3}	1.7×10^{-3}	- 48
Beryllium	4.0×10^{-4}	2.4×10^{-3}	+ 500
1,3-Butadiene	4.6×10^{-7}	2.8×10^{-4}	+ 60770
Cadmium	2.3×10^{-3}	1.8×10^{-3}	- 22
Chloroform	1.0×10^{-5}	2.3×10^{-5}	+ 130
Epichlorohydrin	2.2×10^{-7}	1.2×10^{-6}	+ 445
Ethylene dibromide	5.1×10^{-4}	2.2×10^{-4}	- 57
Ethylene oxide	3.6×10^{-4}	1.0×10^{-4}	- 72
Formaldehyde	6.1×10^{-6}	1.3×10^{-5}	+ 113
Gasoline vapors	7.5×10^{-7}	6.6×10^{-7}	- 12
Methyl chloride	1.4×10^{-7}	3.6×10^{-6}	+ 2,470
Methylene chloride	1.8×10^{-7}	4.7×10^{-7}	+ 161
Nickel (subsulfide)	3.3×10^{-4}	4.8×10^{-4}	+ 45
Perchloroethylene	1.7×10^{-6}	5.8×10^{-7}	- 66
Propylene oxide	1.2×10^{-4}	3.7×10^{-6}	- 97
Styrene	2.9×10^{-7}	5.7×10^{-7}	+ 97
Trichloroethylene	4.1×10^{-6}	1.7×10^{-6}	- 59
Vinyl chloride	2.6×10^{-6}	4.1×10^{-6}	+ 58
Vinylidene chloride	4.2×10^{-5}	5.0×10^{-5}	+ 19

the change has been large (over 100 percent) and, in the case of 1,3-butadiene, the change has been over a 60,000 percent increase.

A third factor that accounts for the apparent increase in estimated cancer risk is a more complete accounting of sources that contribute to cancer risk. As noted above, a potentially significant new source category is TSDFs. Another important source category is electroplating.

The more extensive body of information available to this study has helped provide for a more complete accounting of source categories and pollutants. The apparent increase in estimated cancer risk, therefore, should not necessarily be viewed as a problem that has become worse. Rather, the estimates in this study, which are based on new and more complete information, simply suggest that the problem may be larger than previously thought.

Nature of the Problem

The nature of the air toxics problem can be described in several ways: which pollutants contribute the most to the cancer risk; which sources contribute the most to cancer risk; and how does cancer risk vary from one geographic region to another. Since this study found geographic variations to be of a very similar nature as those reported in the 1985 Six-Month Study, only the first two aspects of the problem will be compared.

Individual Pollutants. For the most part, the same pollutants found to contribute the largest percentages to total annual cancer risk in the 1985 Six-Month Study are also found to be among the larger contributors in the present study. These compounds include hexavalent chromium, PIC, asbestos, benzene, carbon tetrachloride, ethylene dibromide, arsenic, and vinyl chloride.

As seen earlier in Table 3-25, eight of the individual cancer rates (i.e., annual cancer cases per year per million population) calculated for pollutants under the present study fall within the range of cancer rates created by the three studies in the 1985 Six-Month Study. The cancer rates for two pollutants (gasoline vapor and PIC) bound their respective estimates in the 1985 Six-Month Study. This indicates that the magnitudes of incidence for these ten pollutants have been estimated to be approximately the same. For formaldehyde, the cancer rate calculated in this study is lower than that in the Ambient Air Quality study found in the 1985 Six-Month study, but higher than the other two analyses in the 1985 Six-Month Study. The decrease most likely reflects better data and measurement techniques available than were used in the original Ambient Air Quality study.

For the other 10 pollutants identified in Table 3-25, the cancer rates calculated in this study fall outside the range created in the 1985 Six-Month Study. Of these pollutants, four -- ethylene oxide, trichloroethylene, asbestos, and radionuclides -- show a decrease in the estimated cancer rate. For ethylene oxide and trichloroethylene, most of this decrease can probably be attributed to the change in the unit risk factor. For asbestos, the change reflects better emission factors. For radionuclides, a new risk analysis was conducted using updated information on the number of facilities, radionuclide emissions to the air, and control technologies. The net effect of the updated information was a decrease in the estimated risk for radionuclide exposure.

Six pollutants (including dioxin, which was not included in the 1985 Six-Month Study) show an increase in estimated risk. For arsenic, a modest increase in annual cancer cases is estimated (from 60 to 68 per year), which is apparently due to higher measured ambient concentrations.

For ethylene dibromide (EDB) the estimated cancer rate has increased in spite of a decrease in the unit risk factor. This has likely occurred due to the absence of a risk estimate for EDB in the 1985 Six-Month Study from the Ambient Air Quality study. As noted in the present study, modeled estimates appear to underestimate actual ambient concentrations. The present study based the risk estimate on ambient-measured concentrations. Thus, the net effect is an increase in the estimated cancer rate for EDB, with an increase in estimated cancer cases from 27 to 68 per year nationwide.

For both ethylene dichloride and chloroform, the updated Ambient Air Quality study's estimates of cancer risk were selected for the risk estimate. The increase in the estimated cancer cases from chloroform can be attributed in part to an increase in its unit risk factor. For both pollutants, the increase may be simply attributable to a more recent and larger data set that shows higher ambient concentrations than before.

The most dramatic increase is associated with 1,3-butadiene. This has occurred for two reasons. One reason is the increase in the unit risk factor, from 4.6×10^{-7} to 2.8×10^{-4}, an increase of over 600 times. The second reason is that ambient-measured concentrations of 1,3-butadiene were not a part of the 1985 Six-Month Study and the major source of ambient 1,3-butadiene -- motor vehicles -- were not included in the other two studies in the 1985 Six-Month Study. These two factors combined to increase the estimated nationwide cancer risk due to 1,3-butadiene from 0.01 cancer cases per year to almost 270 per year.

Source Categories. In the 1985 Six-Month Study, area and point sources were found each to account for approximately one-half of the aggregate incidence in both the NESHAP and the 35-County studies. When PIC was included (by using BaP as a surrogate), areas sources were found to be dominant, accounting for over 75 percent of the incidence in both the NESHAP and the 35-County studies. This result was noted as being consistent with the fact that PIC was estimated to account for a large portion of aggregate incidence, and that nearly all BaP emissions appear to come from area sources (principally motor vehicles and fuel combustors in small heating units).

Earlier in this chapter, Table 3-11 summarized the estimated contributions of individual source categories to total cancer risk by area vs. point source. Area sources were estimated to contribute approximately 75 percent of the total nationwide annual incidence and point sources, approximately 25 percent. The two studies, thus, show essentially identical estimates of the relative contribution of area vs. point sources in spite of some significantly important pollutants and source categories included in the current study that were not included in the 1985 Six-Month Study.

Table 3-28 presents the results of the Southeast Chicago study in terms of area vs. point and mobile vs. stationary sources. In that study, point sources are estimated to contribute approximately 48 percent of the total estimated annual incidence in the Southeast Chicago area, and area sources approximately 30 percent. (Approximately 20 percent was attributed to background pollutants, the sources of which were not identified.) These relative contributions of area vs. point sources are very different from the nationwide split estimated. The

TABLE 3-28

CONTRIBUTION OF SOURCES TO ESTIMATED ANNUAL CANCER CASES AND AREAWIDE LIFETIME INDIVIDUAL RISKS IN SOUTHEAST CHICAGO

SOURCE TYPE/ CATEGORY	ANNUAL CANCER CASES[a]	AREAWIDE LIFETIME INDIVIDUAL RISK	PERCENT OF TOTAL
Point			
Steel Mills	0.41	7.3×10^{-5}	32
Chrome Platers	0.185	3.3×10^{-5}	15
Other Industrial Sources	0.016	2.9×10^{-6}	1
Sewage Treatment Plants	0.001	1.8×10^{-7}	0.1
Total Point	0.612	1.1×10^{-4}	48
Area			
Home Heating	0.127	2.3×10^{-5}	10
Consumer Sources	0.05	8.9×10^{-6}	4
Mobile Sources	0.22	3.9×10^{-5}	17
Waste Handling	0.001	1.8×10^{-7}	0.1
Total Area	0.398	7.1×10^{-5}	31
Background Pollutants	0.26	4.6×10^{-5}	20
Mobile	0.22	3.9×10^{-5}	18
Stationary	0.79	1.4×10^{-4}	62
Background	0.26	4.6×10^{-5}	20

[a] Southeast Chicago study, p. 33. Values were adjusted to the unit risk factors used in this study.

[b] Calculated by multiplying annual cancer cases by 70 and dividing by population of study area (i.e., 393,000).

larger share attributed to point sources in the Southeast Chicago area is likely due to locally high risk from steel mills.

Mobile sources are also seen to be a relatively lower contributor to total annual incidence versus stationary sources in the Southeast Chicago area. This again is likely due to the locally-high risk from steel mills. In addition, risk from heating appears to be higher than the nationwide estimate, thus further reducing the percent of total annual incidence attributed to mobile sources.

The total areawide lifetime individual risks for the Southeast Chicago study are similar to those reported earlier in this chapter for the Santa Clara study (Table 3-23) and the Kanawha Valley study (Table 3-24). Excluding consideration of "background pollutants," areawide lifetime individual risks from area sources are again in the 10^{-5} range, with mobile sources being the major contributor to areawide lifetime individual risk followed closely by home heating. Point source contribution to areawide lifetime individual risk in Southeast Chicago is higher than the area source contribution, as was seen in the Kanawha Valley study. This seems consistent with the relative nature of the study areas (Southeast Chicago has significant point source contribution from coke ovens.)

4.0 SUMMARY AND CONCLUSIONS

In this chapter, the results of this study are summarized and conclusions are drawn with regard to the magnitude and nature of the cancer problem associated with outdoor exposures to air toxics in the United States. These results are also compared to those contained in the 1985 Six-Month Study.

As has been discussed throughout the report, the results of this study are subject to various limitations and uncertainties. The numerical estimates presented in this report, therefore, should be viewed as rough indications of the magnitude of potential cancer risk caused by a limited group of pollutants found in the ambient air. Many of the absolute values for individual pollutants are almost certainly inaccurate. The best use of these estimates is in describing the broad nature of the cancer risk posed by these toxic air pollutants and by making relative comparisons of risks between pollutants and source categories.

Magnitude of the Cancer Risk

Annual Cancer Incidence

Based on the pollutants and source categories examined, nationwide annual cancer incidence is estimated to be between 1,700 and 2,700 cancer cases per year (see Table 3-1). This is equivalent to between 7.2 and 11.3 cancer cases per year per million population (1986 population of 240 million). Approximately one-third of this cancer risk was attributed to exposure to products of incomplete combustion (PIC). Of all the cancer risks estimated in this study, the greatest degree of uncertainty is mostly likely associated with the cancer risk estimate for PIC.

A range of annual cancer incidence is reported as the result of uncertainties associated with primarily four individual pollutants that also are estimated to be among the largest individual contributors to cancer risk. These four pollutants are PIC, dioxin, gasoline vapors, and hexavalent chromium. The uncertainties identified are associated primarily with: (1) the inability at this time to select a single unit risk factor from a range of unit risk factors for diesel particulates, which are included with PIC; (2) the sampling and extrapolation methodologies for dioxin; (3) the identification of the cancer-causing portion of gasoline vapors; and (4) the portion of total ambient chromium that is hexavalent. Although point estimates were made for most pollutants, the lack of a range does not mean there is no uncertainty associated with the absolute magnitude of the cancer risk estimate.

The 1985 Six-Month Study presented three separate analyses that showed a range of cancer rates from approximately 5 to 7.4 cancer cases per year per million population. The results of the current study estimated a cancer rate of between 7.2 and 11.3 cancer cases per year per million population (see Table 3-25). Using a total 1986 U.S. population of 240 million, the results of this study show approximately 500 to 900 more cancer cases per year (comparing lower and upper ranges). This "increase" does not necessarily indicate a growing problem, but is more likely the result of analysis of more air toxic pollutants than were considered in the 1985 Six-Month Study and, in some instances, a better accounting of sources (e.g., sources that emit 1,3-butadiene). Even though this study has a broader data base to draw upon than was available to the 1985 Six-Month Study, it is recognized that cancer risk estimates are being made for a only a portion of total ambient air pollutants and for a portion of all sources. In addition, quantitative risk estimates from pollutants formed or transformed in the atmosphere (secondary formation) remain unquantified for almost all pollutants. Evidence to date suggests secondarily-formed pollutants may pose a significant component of total cancer risk. Based on these considerations, the actual magnitude of the problem, therefore, can easily be larger than estimated in this study. On the other hand, the estimates presented in this study are based on the use of unit risk factors that are either upper-bound estimates or maximum likelihood estimates of the carcinogenicity of a pollutant. Quantitative estimates derived from the use of these unit risk factors, therefore, could overstate the true risk from a pollutant. The net effect of these and other uncertainties (e.g., assessing exposures) on total risk is unknown. It is expected, nevertheless, that the pollutants and source categories considered herein are among the major contributors to cancer risk from air toxics based on our current state of knowledge.

Lifetime Individual Risks

Maximum lifetime individual risks of 1 x 10^{-4} (1 in 10,000) or greater were reported in almost all of the studies examined for this report (see Table 3-2). Risk levels this high were reported for such specific sources as major chemical manufacturers, waste oil incinerators, hazardous waste incinerators, publicly owned treatment works (POTWs), steel mills, hospitals, traffic intersections, and hazardous waste treatment, storage, and disposal facilities (TSDFs).

Risk levels reported for areawide lifetime individual risks, which includes risk from point and area sources, were generally around 10^{-4} (see Table 3-6).

On an individual pollutant basis, maximum individual risks of 1×10^{-4} or greater were reported for 16 of the pollutants included in the NESHAP/ATERIS data base (see Table 3-2). Twelve of these pollutants were estimated to have maximum individual risks of 1.0×10^{-3} or greater. These estimates of risks are related to specific sources. However, because of the nature of the assessments contained in the ATERIS data base, there is a very large degree of uncertainty associated with some of these estimates for specific sources.

Multi-pollutant lifetime individual risks in four urban areas due to exposure to 9 to 16 pollutants (at one monitoring site in each urban area) ranged from 3×10^{-4} to 3×10^{-3} (see Table 3-7). These estimates were based on ambient-measured data and generally cannot be related to specific point sources.

While the present study shows the estimate of nationwide cancer cases to be somewhat larger than was estimated in the 1985 Six-Month Study, the maximum and areawide lifetime individual risks estimated in the present study are nearly identical to those estimated in the 1985 Six-Month Study. The broader scope of the present study has resulted in identifying additional types of sources (e.g., TSDFs, POTWs) that can contribute to significant maximum individual risks.

Nature of the Cancer Risk

Individual Pollutants

As discussed in Chapter 2, there is considerable uncertainty with the absolute risk estimates for some of the pollutants examined in this study. Nevertheless, the available information indicates seventeen[1] of the approximately 90 pollutants examined may each account for 10 or more cancer cases per year nationwide. Of these, thirteen may each account for 40 or more cancer cases per year. These thirteen are: PIC; 1,3-butadiene; hexavalent chromium; formaldehyde; benzene; chloroform; asbestos; dioxin; arsenic; ethylene dibromide; gasoline vapors; ethylene dichloride; and carbon tetrachloride.

The seventeen compounds that are estimated to contribute at least 10 excess cancer cases per year nationwide appear to be most frequently associated with high maximum individual risks. However, other compounds may be the most significant contributor to the maximum individual risk for a particular city. For example, coke oven emissions in the Southeast Chicago study contributed over 75 percent of the highest estimated lifetime individual risk. Individual compounds, such as epichlorohydrin and styrene, that have small aggregate cancer incidences may also be associated with high maximum individual risks (greater than 1×10^{-4}).

For the most part, the individual compounds found to be the more important contributors to cancer risk in the present study are the same as those found in the 1985 Six-Month Study. The most significant difference is the addition of 1,3-butadiene to the list of potentially important contributors. Dioxin may also be a significant contributor, but the uncertainties associated with its risk estimates make it difficult to conclude this at this time. Several pollutants, on the other hand, appear to be somewhat less of a factor in terms of aggregate cancer risk, but not necessarily in terms of maximum individual risk. Changes in the pollutants identified in the present study and in the 1985 Six-Month Study as the more important contributors are primarily due to the broader scope of the present study and to newer estimates of the unit risk factors for the individual pollutants.

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[1] Acrylonitrile, arsenic, asbestos, benzene, 1,3-butadiene, cadmium, carbon tetrachloride, chloroform, chromium (hexavalent), dioxin, ethylene dibromide, ethylene dichloride, formaldehyde, gasoline vapors, PIC, vinyl chloride, and vinylidene chloride.

Sources

As in the 1985 Six-Month Study, a wide variety of sources contribute to aggregate incidence and individual risk (see Table 3-11). Motor vehicles were found to be the largest contributor to nationwide annual incidence, contributing almost 58 percent of total incidence (including estimated risk attributable to the secondary formation of formaldehyde). The risk associated with the secondary formation of formaldehyde was estimated to account for 6.5 percent of the total estimated incidence (130 annual cancer cases). Of these 130 annual cancer cases, 93 are estimated to be attributable to volatile organic compound (VOC) emissions from area sources (including 45 from mobile sources) and 37 from point sources (see Table 3-10). Electroplating (6%) was the third largest contributor to aggregate incidence as a result of chromium emissions. The next five major contributors were TSDFs (5%); woodsmoke (5%); asbestos, demolition (4%); gasoline marketing (3%); and solvent use/degreasing (3%). Unspecified point sources (3%) and cooling towers (3%) were the ninth and tenth largest contributors to total annual incidence.

In general, a significant portion of the cancer risk from specific sources was usually due to a few pollutants, even where a source emitted many different pollutants. For example, over 70 pollutants were included in the analysis on hazardous waste combustors, but two pollutants (cadmium and hexavalent chromium) were estimated to be responsible for almost 90 percent of the estimated cancer cases from hazardous waste incinerators and three pollutants (cadmium, hexavalent, chromium, and arsenic) for almost 90 percent of the estimated cancer cases from hazardous waste boilers and furnaces.

Both mobile and stationary sources were found to contribute significantly to total nationwide annual incidences. Considering both direct emissions to the atmosphere and secondary formation of formaldehyde, mobile sources were estimated to contribute approximately 58 percent and stationary sources approximately 42 percent of total annual incidence. Area sources were found to contribute approximately 75 percent and point sources approximately 25 percent of the total cancer incidence (see Table 3-11).

The relative contribution of the aggregate types of sources (i.e., point vs. area, mobile vs. stationary) to total annual incidence can vary significantly for specific geographic areas. For example, the Southeast Chicago study showed point sources contributing almost 50 percent (vs. the 20 percent noted above) and stationary sources approximately 60 percent (vs. 42 percent from above) of the total annual incidence estimated for Southeast Chicago (see Table 3-28). These differences are most likely due to the significant contribution to risk from steel mills in the Southeast Chicago area.

With regard to lifetime individual risk, reported maximum individual risks usually were associated with specific point sources, such as industrial facilities or chemical manufacturers. Based on the information in the IEMP-Santa Clara study, the levels of maximum individual risk associated with individual area-type sources (e.g., gasoline marketing, degreasers, waste oil burning) appear to be lower than those found for sources typically included in a point source category (see Table 3-22). However, the IEMP-Santa Clara study found a maximum lifetime individual risk of 10^{-4} for at least one traffic intersection. On the other hand, not all point sources have high maximum lifetime individual risks associated with them. In fact, the majority of point sources in some source categories have maximum individual risks of 10^{-6} or less.

As noted earlier, areawide lifetime individual risks were generally lower than the maximum individual risk values within comparable geographic locales. The relative contribution of area and point sources to areawide lifetime individual risks can vary from one locale to another. For example, the IEMP-Santa Clara showed area sources contributing approximately 50 percent of the areawide lifetime individual risk and point sources approximately 15 percent (see Table 3-23). (The remaining 25 percent was from carbon tetrachloride, which was not allocated in that study to either area or point source.) In contrast, the Southeast Chicago study shows point sources contributing approximately 48 percent and area sources approximately 31 percent of the areawide lifetime individual risk (the remaining 20 percent was from formaldehyde and carbon tetrachloride, which were not allocated to either area or point source) (see Table 3-28).

Among area sources, mobile sources were found to be responsible for between 50 and 60 percent of the areawide lifetime individual risk (see Tables 3-23, 3-24, and 3-28). Solvent use and heating in the IEMP-Kanawha Valley study (see Table 3-24) and home heating in the Southeast Chicago study (see Table 3-28) were identified as having areawide lifetime individual risks approximately one-half to one-quarter as large as those associated with mobile sources.

Geographic Variability

Exposure to individual air toxics varies on a city-to-city basis as well as on an intra-city basis. For some pollutants, such as benzene, the variation appears to be relatively small, less than a factor of two (see Table 3-13). For other pollutants, the variation is higher, ranging to a factor of almost 20. For the pollutants compared, the degree of variation in ambient concentrations for a particular pollutant apparently can vary by the same degree within a city as between cities.

The variations in ambient concentrations for individual pollutants can lead to variations in the number of cancer cases and the cancer rate (i.e., cancer incidence per year per million population) between geographic areas. In spite of the differences in risk attributable to individual pollutants, areawide lifetime individual risks were found to be generally the same between the geographic locales examined in this study (see Table 3-6). Particular geographic locales may have substantially higher areawide lifetime individual risk. If this occurs in a relatively sparsely populated locale, a low absolute number of cancer cases would mask a high cancer rate and this higher-than-average areawide lifetime individual risk. In a similar manner, a relatively low areawide lifetime individual risk may mask a significant maximum individual risk that affects a small portion of the local population.

Most of the geographic locales reviewed in this study showed comparable maximum or highest estimated lifetime individual risk levels (see Table 3-15). However, this does not mean that the same number of people are exposed to that level of risk in each city.

The pollutants and source categories that are the most important contributors to risk (annual incidence and maximum individual risk) in a geographic area will vary from one area to another. For some pollutants, the variation may be relatively small and the primary source will be the same between areas. For example, benzene was found to contribute between approximately 5 and 10 percent of the total annual incidence in six cities, with between 45 and 80 percent of the benzene-related cancer incidence attributed to motor vehicles (see Table 3-17). Other pollutants show a wider range of variation, and the relative contribution for some pollutants can be dramatically affected by the presence of major point sources. For example, in five of the six selected cities, 1,3-butadiene was estimated to contribute between 6 and 24 percent of the total cancer incidence, all attributable to motor vehicles. In the sixth city, over 48 percent of the total cancer incidence was attributed to 1,3-butadiene. Of the 1,3-butadiene-related cancer incidence in this city, over 80 percent was attributed to chemical manufacturing plants and less than 20 percent to motor vehicles.

In general, the results and conclusions of the present study are consistent with those drawn in the 1985 Six-Month Study regarding geographic variability.